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H. Preston Hoskins, Secretary-Editor, 537 Book Building, Detroit, Mich.

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THE 1931 CONVENTION CITY

For the third time in approximately a quarter of a century Kansas City, Missouri, this year will entertain the American Veterinary Medical Association. The first such occasion was in 1907. The second time was ten years later, in 1917. On both occasions the Kansas City Veterinary College was in operation, and this well-known institution was the center of convention activities. This year our meeting will be held without the facilities which are usually found in connection with a large educational institution. Although not absolutely essential, they will be missed, especially in connection with the clinic. Possibly sensing this situation, the Local Committee on Arrangements has gone to unusual lengths to provide an outstanding clinic this year, and we have no reason to doubt that these elaborate plans will materialize.

It is customary, each year, to say something in the JOURNAL about the history and attractions of the convention city. It could hardly be expected that the editor of a veterinary journal could improve upon the work of a convention bureau manager in describing the attractions of his city. Accordingly, we have drawn rather fully from some material supplied us by Mr. W. M. Symon, manager of the Kansas City Convention Bureau.

A trifle more than one hundred years ago an Indian scout, riding along the bluffs that overlook the valley where the Kaw River joins the Missouri, saw a thin curl of smoke rising from a new log cabin. To the Indian scout this merely meant that a white trapper had pushed farther west than his fellows.

Could the scout have peered down through a century, the log cabin would have been lost in the smoke of a thousand factories; the clatter of stones dislodged by his horse's hoofs would have been drowned by the increasing hum of industry's wheels and the woods and grassy meadows would have been covered by

KANSAS CITY FROM THE AIR

The heart of the business district of the "Heart of America," where the 68th Annual A. V. M. A. Convention will be held in August

one of the most beautiful residential and park districts in the world.

The big fur companies were quick to see the advantage of this section. The fur trade flourished up to 1819, then diminished. Next came the agricultural pioneer. Where wild grasses had grown there appeared grains of all kinds. Ranchers and stockmen supplanted hunters and trappers. Closely following the settler came the industrial pioneer. The dealers in plows and household goods erected their log shacks.

Steamboats had been running on the Missouri River since 1819. Independence was founded in 1827. In 1833, the town

of Westport, now part of Kansas City, was established. These pioneer villages became outfitting posts for the caravans that crossed the prairies to the new West.

In 1839, the town of Kansas was founded on the river banks, and it quickly overshadowed the other two. The name came from the Kahn or Kanzas Indians, not from the state of that name, as all the territory to the West was then known as the Nebraska Territory. The state was named later.

The city was incorporated as the "Town of Kansas" in 1850. This was changed to the "city of Kansas" in 1853, and to "Kansas City" in 1889. The first post office was established in 1845, and the first newspaper in 1851. The telegraph appeared in 1858, followed by the first railroad in 1864. A bridge was built across the Missouri River in 1869, and street railways made their appearance the following year.

It was at Kansas City that the famous Santa Fe Trail began. Near here Civil War battles raged, each side seeking control of the West. The fiercest battle of the campaign, giving the North control, was fought near Westport.

In the era of the open range, great droves of cattle were herded from the prairies to western rail terminals and thence hauled into Kansas City. This founded the great live stock and packing house enterprise here, financing and merchandising institutions developed and industries took a foothold.

Kansas City today is a homey and hospitable place for resident, visitor or convention delegate. It truthfully is said of Kansas City: "It blends the culture of the East, the vision of the West, the energy of the North, and the hospitality of the South."

If you attended either of the previous conventions held in Kansas City, you will no doubt want to go again this year. If it was not your privilege to attend either of these previous conventions, it is just as probable that you will want to go to Kansas City and see the kind of convention that is held there. We hope to see you in the "Heart of America," August 25-26-27-28.

A GLIMPSE OF THE KANSAS CITY PROGRAM

Through the splendid coöperation of the secretaries of the five sections, we are able to give our members a glimpse of the literary program of the Kansas City meeting. We are merely listing the titles of the papers thus far secured, with the names of their authors, in order to give some idea of the ground that will be

covered by these papers at the meeting. Considerable rearrangement will be necessary before the program is in final form. It is hoped that we will be able to publish the complete program of the meeting in the August issue of the JOURNAL.

World-Wide Prevalence of Infectious Live Stock Diseases—Dr. George W. Pope, U. S. Bureau of Animal Industry, Washington, D. C.

Studies in Idiopathic Prenatal Anemia of Young Pigs—Drs. Robert Graham, Frank Thorp, Jr., and W. A. James, University of Illinois, Urbana, Ill.

Foot-and-Mouth Disease and Vesicular Stomatitis—Dr. Kurt Wagener, Berliner Tierarztliche Hochschule, Berlin, Germany.

Hog-Lot Sanitation and Its Relation to Swine Disease Control—Dr. H. A. Wilson, State Veterinarian, Jefferson City, Mo.

Tick Eradication—Dr. Joe H. Bux, State Veterinarian, Little Rock, Ark.

Anthrax—Dr. N. F. Williams, State Veterinarian, Fort Worth, Texas.

Problems of Tuberculosis Control and Its Relation to Sanitary Science and Food Hygiene—Dr. Peter Malcolm, State Veterinarian, Des Moines, Iowa.

Sheep Diseases—Dr. Hadleigh Marsh, Montana Veterinary Research Laboratory, Bozeman, Mont.

Bang's Disease (Contagious Abortion) in Cattle and Its Relation to Undulant Fever in Man—Dr. Charles H. Kitselman, Kansas State College, Manhattan, Kans.

Spontaneous Infection with *Brucella Abortus* in the Bull—Drs. F. B. Hadley and E. B. Osborn, University of Wisconsin, Madison, Wis.

Clinical Studies on Retained Placenta in Cows—Dr. C. C. Palmer, University of Delaware, Newark, Del.

Nutritional Deficiency in Its Various Forms—Dr. A. F. Schalk, Ohio State University, Columbus, Ohio.

The Value of Iodin in Cattle Practice—Dr. E. C. McCulloch, University of Wisconsin, Madison, Wis.

Post-Vaccination Troubles in Swine—Dr. Henry Hell, Wilton Junction, Iowa.

Sheep Diseases and Experimental Studies—Dr. Henry W. Turner, Pennsylvania Bureau of Animal Industry, Harrisburg, Pa.

Care of the Ewe Before and After the Lambing Period—Dr. L. A. Hammers, Clearwater, Kans.

Feeding Colorado Lambs—Dr. I. E. Newsom, Colorado Agricultural Experiment Station, Fort Collins, Colo.

County-Wide Eradication of Equine Parasites—Dr. John B. Bryant, Mount Vernon, Iowa.

Some Bipolar Organisms Found in Pneumonia in Sheep—Drs. I. E. Newsom and Floyd Cross, Colorado Agricultural Experiment Station, Fort Collins, Colo.

Report of Further Work on the Relation of *Bacterium Abortus* Bang to Fistula and Poll Evil of Horses—Dr. C. P. Fitch and Lucille Bishop, University of Minnesota, Saint Paul, Minn.

The Relation of the Time Element to the Results Obtained by the Rapid Agglutination Test for Bang's Disease—Dr. C. R. Donham, University of Minnesota, Saint Paul, Minn.

Blackleg Immunization—Dr. J. P. Scott, Kansas State College, Manhattan, Kans.

Preliminary Bacteriological Report on Shipping Fever—Dr. J. P. Scott, Kansas State College, Manhattan, Kans.

Comparative Germicidal Tests of Mercurochrome and Tincture of Iodin—Maj. R. A. Kelser and Lt. R. W. Mohri, Army Medical School, Washington, D. C.

Studies on Canine Distemper. I. The Bacteriology of One Hundred Naturally Infected Cases—Dr. A. S. Schlingman, Detroit, Mich.

A Modification of the Rapid Agglutination Test for Pullorum Disease—Dr. Howard Welch, Montana Agricultural Experiment Station, Bozeman, Mont.

The Resistance of the Bacillus of Johne's Disease to Lytic Influences—Dr. W. A. Hagan and Eveoleen Rexford, New York State Veterinary College, Ithaca, N. Y.

Non-Parasitic Skin Diseases of Dogs—Dr. E. C. Khuen, Chicago, Ill.

Treatment of the Diseases of the Eye and Accessory Organs—Dr. D. A. Eastman, Moline, Ill.

The Treatment of Fractures—Dr. E. B. Dibbell, Baltimore, Md.

Meningo-Cerebral Complications in Canine Distemper—Drs. Ashe Lockhart and S. R. Johnson, Kansas City, Mo.

Feeding Dogs—Dr. C. F. Schlotthauer, Mayo Foundation, Rochester, Minn.

Diseases of Foxes—Dr. Karl B. Hanson, U. S. Fur Farm Experiment Station, Saratoga Springs, N. Y.

Anesthetics for Small Animals—Their Indications and Uses (Illustrated)—Dr. E. R. Frank, Kansas State College, Manhattan, Kans.

Some Common Diseases of Cats—Dr. H. K. Miller, Mamaroneck, N. Y.

The Hatchability of Eggs and the Livability of Chicks of Pullorum-Infected and Non-Infected Hens—Dr. H. C. H. Kernkamp, University of Minnesota, Saint Paul, Minn.

Studies of Leukemia of Fowls—Dr. R. Fenstermacher, University of Minnesota, Saint Paul, Minn.

Tuberculosis of Turkeys, with Special Reference to Tuberculin Testing—Dr. W. R. Hinshaw, University of California, Davis, Calif., Dr. K. W. Niemann, University of Nevada, Reno, Nevada, and Dr. W. H. Busic, Chico, Calif.

Studies of Some Virus Diseases of Fowls—Drs. C. A. Brandly and L. D. Bushnell, Kansas State College, Manhattan, Kans.

Observations on Prolapse or So-called "Blow Out" in Chickens —Dr. H. J. Stafseth, Michigan State College, East Lansing, Mich.

Comparison of Three Methods of Testing for Pullorum Disease, with Finer Interpretations of Readings on the Old Tube Agglutination Test—Dr. A. J. Durant, University of Missouri, Columbia, Mo.

Some Observations on Duck Diseases—Dr. H. J. Stafseth, Michigan State College, East Lansing, Mich.

IOWA VETERINARIANS REDUCE FEES

Iowa veterinarians will charge less, this year, for immunizing swine against hog cholera, than they have charged in recent years, according to a survey conducted by the Iowa Veterinary Medical Association. Part of the reduction is made possible by the lower prices prevailing for anti-hog cholera serum. The balance of the cut will be borne by the veterinarians, as their contribution to the cause of sound live stock sanitation. The market price of hogs is down. This makes cheaper serum possible, but it also tends to cause farmers to refrain from adding the cost of immunization to the other items entering into the cost of pork production. It is to be hoped that the lowering of the expense of vaccination will stimulate the widespread use of serum and virus, thereby insuring the best possible protection against the most serious scourge of the swine industry.

PROPER MATERIAL FOR FARM PAPERS

In the issue of *The Indiana Farmers Guide* for May 23, 1931, there was a very timely article from the pen of Dr. R. A. Craig, of Purdue University, entitled, "When Animals Get Sick." Two very important points were brought out by Dr. Craig, namely, the value of a correct diagnosis before a disease can be treated intelligently, and the necessity for research work with animal diseases before these can be treated or controlled successfully. Articles such as this one, appearing in farm papers even only occasionally, will accomplish much more good than the old-fashioned, veterinary, question-and-answer column, with its long-distance diagnoses and shot-gun prescriptions, as a regular weekly feature.

EXECUTIVE BOARD ELECTIONS

The forms of this issue of the JOURNAL have been held open long enough for us to be able to announce the results of the elections held in Executive Board Districts 6 and 8, which came to a close on June 27.

In District 6, Dr. Leslie M. Hurt, of Los Angeles, Calif., was a winner by a very slight margin over Dr. J. P. Iverson, state veterinarian of California.

In District No. 8, Dr. N. F. Williams, state veterinarian of Texas, was an easy winner over the four other candidates on the ticket. Dr. Williams was elected to the Board for a one-year term in 1930. Both Dr. Hurt and Dr. Williams were elected for full terms of five years. Dr. Hurt succeeds Dr. Geo. H. Hart, of Davis, Calif., who was not a candidate for reëlection.

Doctor McInnes Makes First Contribution

Dr. Benjamin McInnes, of Charleston, South Carolina, under date of May 23, 1931, forwarded his check for five dollars to Dr. Adolph Eichhorn, chairman of the A. V. M. A. Special Committee on International Veterinary Congress, as a contribution toward the fund to be raised for defraying the expenses of entertaining the 1934 Congress. We understand that this is the first actual contribution for the purpose, although several state and local veterinary associations have taken steps to do their share, in a financial way, toward making the Congress a success.

APPLICATIONS FOR MEMBERSHIP

Although the number of applications for membership received during the first six months of 1931 fell below the number for the corresponding period of 1930, the results of the first six months this year are in no way disappointing. As a matter of fact, the number of applications filed has been very gratifying for the reason that only one of our resident secretaries has put on a systematic membership campaign.

One of the high spots of the past month was the receipt of fifteen applications from members of the graduating class at the Ohio State University, obtained through the efforts of Dr. W. F. Guard, our resident secretary for Ohio and an indefatigable worker for the A. V. M. A. The one membership campaign put on this year was in Kansas, under the direction of Dr. E. J. Frick, of Kansas State College, whose efforts yielded fourteen applications from Kansas and adjoining states.

Six months of 1931 remain. There is plenty of time to pass the 1930 figure of 252 applications. Look around and see if all the veterinarians in your immediate vicinity are members. If you find one who is not, send his name and address to the Secretary and request that an application be sent the prospective member. Quite a large number of the applications received this year have resulted from personal solicitation upon the part of members. Ten new members can be obtained in this way, while one is being secured by letter-writing.

Keep in mind the manner of handling applications. Here are the regulations:

> Applications for membership shall be made upon blanks furnished by the Association, in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Applications must be accompanied by a membership fee of $5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Applications must be filed with the Secretary and examined by him for correctness and completeness as far as available information will allow. After such approval by the Secretary, the latter will cause to be published in the official JOURNAL, as soon thereafter as possible, said application with name and address of the applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the Secretary, as against the applicant being admitted to membership in the Association, his name shall again be listed in the next issue of the JOURNAL, and if no objections shall have been filed within thirty days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the Secretary and membership card issued. If any objections be filed against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

FIRST LISTING

BAILEY, LEONARD PERRY — South Charleston, Ohio
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

BENNER, KENNETH LELAND — Bainbridge, Ohio
D. V. M., Ohio State University, 1931
Vouchers: Oscar V. Brumley and W. F. Guard.

BENNETT, PAUL CLIFFORD — 288 E. 12th Ave., Columbus, Ohio
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

BLOOM, FRANK — 37-19 Junction Ave., Corona, L. I., N. Y.
D. V. M., Cornell University, 1930
Vouchers: Peter Olafson and W. A. Hagan.

BUEHLER, HAROLD JOHN — Saint Edward, Nebr.
D. V. M., Michigan State College, 1929
Vouchers: Frank Breed and Floyd Perrin.

BURRISS, JAMES THOMAS — Smithfield, Ohio
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and Walter R. Krill.

CAMPBELL, MACFARLAND — 601 W. B St., McCook, Nebr.
D. V. S., Kansas City Veterinary College, 1911
Vouchers: Roy V. Loudon and Walter R. Anderson.

CASLICK, FREDERICK GEORGE — Box 45, Versailles, Ky.
D. V. M., Cornell University, 1930
Vouchers: Robert H. Bardwell and Edward A. Caslick.

CROSBIE, GLEN GAROLD — 1115 E. Van Buren St., Phoenix, Ariz.
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

DRUMM, ORVILLE HERRICK — 8518 109th St., Richmond Hill, L. I., N. Y.
V. M. D., University of Pennsylvania, 1930
Vouchers: H. E. Bemis and Alexander Slawson.

FENNER, WALTER HARMON — 821 W. Main St., Hillsboro, Ohio
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

GEURKINK, WALTER — 50 E. Buchtel Ave., Akron, Ohio
D. V. M., Kansas State College, 1931
Vouchers: E. E. Leasure and C. A. Brandly.

HANCOCK, AMOR E. — 2147 Neil Ave., Columbus, Ohio
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

HARDY, W. T. — Substation 14, Sonora, Texas
D. V. M., Agricultural and Mechanical College of Texas, 1930
Vouchers: R. P. Marsteller and N. F. Williams.

HODGES, HARRY G. — 330 N. Van Dien Ave., Ridgewood, N. J.
D. V. M., Cornell University, 1916
Vouchers: F. D. Holford and E. L. Wilson.

HOLLOWAY, SYDNEY HAWTREY — 711 Boyd Bldg., Winnipeg, Man.
B. V. Sc., Ontario Veterinary College, 1929
Vouchers: J. L. Trudeau and C. J. Johannes.

KUBIN, EDISON F. — McPherson, Kans.
D. V. M., Kansas State College, 1909
Vouchers: Edwin J. Frick and R. R. Dykstra.

MCPHERSON, JAMES E. — 308 E. 10th St., Okmulgee, Okla.
D. V. M., Indiana Veterinary College, 1915
Vouchers: C. R. Walter and O. E. Robinson.

MIARS, GRANVILLE JOHN — Jackson Center, Ohio
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

MOLINE, ERNEST N. — 135 N. Concord St., South Saint Paul, Minn.
D. V. S., Kansas City Veterinary College, 1911
Vouchers: G. E. Totten and M. E. Schwab.

MUELLER, WILLIAM KARL 1524 Grayson St., San Antonio, Texas
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

NANKERVIS, THOMAS PRESOW 918 E. Camp St., Ely, Minn.
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

PASTORS, CHARLES ROBERT Rayland, Ohio
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

RICHARDSON, LEONARD ROBERT R. F. D. 8, Box 35, Akron, Ohio
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

SIMMONS, HENRY C. Brookhaven, Miss.
D. V. M., Terre Haute Veterinary College, 1918
Vouchers: G. B. Bradshaw and E. H. Durr.

SMILEY, H. DEVORE Waynoka, Okla.
D. V. M., Kansas State College, 1930
Vouchers: Chas. H. Kitselman, T. J. Leasure and C. C. Hisel.

SNYDER, JESSE B. 1419½ N. Federal Ave., Mason City, Iowa
M. D. V., McKillip Veterinary College, 1911
Vouchers: C. E. Mootz and S. L. Ries.

STAM, EDWARD L. 1632 W. Roosevelt St., Phoenix, Ariz.
B. S., D. V. M., State College of Washington, 1920
Vouchers: J. C. McGrath and H. E. Pine.

STATES, CARL S. Wilmington, Ohio
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

VOLKMAR, FRITZ 39 W. Frambes Ave., Columbus, Ohio
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

WELLS, JOHN LYLE Blue Springs, Mo.
D. V. M., Kansas City Veterinary College, 1915
Vouchers: A. T. Kinsley and H. A. Wilson.

WENGER, RAYMOND DEAN R. 2, Galena, Ohio
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

Applications Pending

SECOND LISTING

Barstow, Ivan L., Box 309, Moscow, Idaho.
Boone, Ralph Wesley, DeSoto, Kans.
Catlett, James Garland, 2418 N. Miami Ave., Miami, Fla.
Crawford, Andy, Heidelberg Hotel, Jackson, Miss.
Elson, R. E., Vinton, Iowa.
Guinn, Cloyde L., Dome of Federal Bldg., Saint Louis, Mo.
Hoffmaster, William Dean, 321 S. 4th Ave., South Saint Paul, Minn.
Hoover, Earl Fremont, 4328 Walnut St., Kansas City, Mo.
Hurtig, Victor Carl, Courtland, Kans.
Koll, Harry, 2017 Texas St., El Paso, Texas.
Khuen, Edward Charles, 2366 Milwaukee Ave., Chicago, Ill.
Luckeroth, Joseph Clemence, Seneca, Kans.
Quist, David Gunnard, Ogden, Iowa.
Riester, Frank H., Buechel, Ky.
Smith, Geo. I., Box 454, Sedan, Kans.
Thomson, William Maxwell, 37 Atlantic St., Salamanca, N. Y.
Tice, Harry R., Summerfield, Kans.

The amount which should accompany an application filed this month is $7.50, which covers membership fee and dues to January 1, 1932, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

San Diego-Imperial Veterinary Medical Association. San Diego, Calif. July 1, 1931. Dr. A. P. Immenschuh, Secretary, Santee, Calif.

Minnesota State Veterinary Medical Society. University Farm, Saint Paul, Minn. July 2-3, 1931. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

North Dakota Veterinary Medical Association. Fargo, N. Dak. July 6-7, 1931. Dr. Lee M. Roderick, Secretary, N. Dak. Agr. Coll., State College Station, Fargo, N. Dak.

Maine Veterinary Medical Association. Bangor, Me. July 8, 1931. Dr. L. E. Maddocks, Secretary, R. F. D. 2, Augusta, Me.

Kentucky Veterinary Medical Association. Seelback Hotel, Louisville, Ky. July 8-9, 1931. Dr. J. R. Stifler, Secretary, Lebanon, Ky.

Tulsa County Veterinary Association. Tulsa, Okla. July 9, 1931. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Virginia State Veterinary Medical Association. Richmond, Va. July 9-10, 1931. Dr. I. D. Wilson, Secretary, Virginia Polytechnic Institute, Blacksburg, Va.

New Jersey, Veterinary Medical Association of. Monterey Hotel, Asbury Park, N. J. July 9-10, 1931. Dr. John G. Hardenbergh, Secretary, c/o Walker-Gordon Lab. Co., Plainsboro, N. J.

Kansas City Association of Veterinarians. Baltimore Hotel, Kansas City, Mo. July 14, 1931. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

Chicago Veterinary Medical Association. Atlantic Hotel, Chicago, Ill. July 14, 1931. Dr. C. L. Miller, Secretary. 508 S. Humphrey Ave., Oak Park, Ill.

South Carolina Association of Veterinarians. Charleston, S. C. July 14-15, 1931. Dr. G. J. Lawhon, Secretary, Hartsville, S. C.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. July 15, 1931. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.

Northwestern Ohio Veterinary Association. Maumee River Yacht Club, Toledo, Ohio. July 16, 1931. Dr. W. P. S. Hall, Secretary, Ch. Food & Drugs, Division of Health, Toledo, Ohio.

Western New York Veterinary Medical Association. Webster, N. Y. July 16, 1931. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

Montana Veterinary Medical Association. Bozeman, Mont. July 22-23, 1931. Dr. Hadleigh Marsh, Secretary, Agr. Exp. Sta., Bozeman, Mont.

Northwest Veterinary Medical Association. Corvallis, Ore. July 28-30, 1931. Dr. Clifford Ackley, Secretary, Winlock, Wash.

Connecticut Veterinary Medical Association. Norwalk, Conn. August 5, 1931. Dr. Edwin Laitinen, Secretary, 993 N. Main St., West Hartford, Conn.

Hudson Valley Veterinary Medical Society. Kenosha Lake, N. Y. August 12, 1931. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

American Veterinary Medical Association. Baltimore Hotel, Kansas City, Mo. August 25-28, 1931. Dr. H. Preston Hoskins, Secretary, 537 Book Bldg., Detroit, Mich.

Broadcasting Program

The Committee on Broadcasting of the New York State Veterinary Medical Society has announced the following schedule for July:

July 1—"Odors and Flavors in Milk—Causes and Control," Dr. C. L. Kern, Dairymen's League Co-operative Association, Inc., Middletown.

July 8—"When the Cows Come Home," Dr. John McCartney, Borden's Farm Products Company, New York.

July 15—"Minimizing Replacements in Dairy Herds," Dr. Irving O. Denman, former president of the Hudson Valley Veterinary Medical Society, Middletown.

July 22—"Disinfection of Stables," Dr. L. L. Parker, former president of the Hudson Valley Veterinary Medical Society, Catskill.

These broadcasts are now given on Wednesday of each week from 12:20 to 12:30 p. m., E. S. T., from Station WGY, Schenectady, N. Y.

Missouri Valley Meeting

Dr. E. R. Steel, secretary of the Missouri Valley Veterinary Association, has announced that there will be no meeting of the organization this summer, in deference to the American Veterinary Medical Association convention in Kansas City. The 1932 summer meeting will be held in Omaha, Nebraska.

ETHICS IN VETERINARY MEDICINE

By MAURICE C. HALL, *Washington, D. C.*

President, American Veterinary Medical Association

The subject of ethics is not a simple subject. Ethical concepts have varied with the background and environment of different races at different places and in different times. They are subjective concepts, not objective things subject to measurement and analysis. As subjective concepts they are matters influenced by early impressions and teachings and are incorporated in our patterns of behavior in childhood through the moulding influence of example and precept.

An attempt to formulate a code of ethics for a profession presents the obvious difficulties which arise from the fact that while outward acts are more or less significant of inward motives, the bald statement of an act as ethical or unethical does not give adequate consideration to the motives and the qualifying facts of the specific circumstances surrounding an act. In practice we actually judge the acts of persons in connection with our knowledge of their previous conduct, our estimate of their general ethical level, and our knowledge of the circumstances leading to and accompanying the acts. The code of ethics is the ritualistic, dogmatic and ceremonial aspect of our recognition of right or wrong conduct, but back of it and in our application of it there must be the concept of the spirit of our ethics.

In effect a code of ethics is like a code of laws—the observance of the code in either case does not ensure that wrong doing is automatically obviated, nor does the violation of the code in either case ensure that wrong doing automatically follows. It is common knowledge that many persons do evil within the law, and that law-breakers sometimes do good by breaking the law. Mankind regards the participants in an unsuccessful revolution as traitors, and the participants in a successful revolution as patriots. If life is more valuable than traffic regulations, a physician need have no scruples about driving faster than the prescribed speed limits when a life is at stake.

In spite of the difficulties involved in formulating a code of ethics, we are not likely to overestimate the importance of ethical conduct, and we must concede the desirability of formulating the specific things which are agreed on as ethical or unethical.

The specification of these things has value in our individual lives, and has usefulness in ascertaining whether a professional organization should accept or retain members otherwise qualified but ethically undesirable. These codes should represent at least minimum essentials necessary for uniformity in establishing standards for professional men.

Admitting that we must have a code and that it presents difficulties in formulation, interpretation and application, it is worth while to examine our code from time to time, to refresh our memory as to its provisions, to consider its limitations, and perhaps to modify it. Such a procedure is only a recognizion of the fact that human instruments must be imperfect and that we must always aim at improvement. The great Jefferson felt that the Constitution of the United States should be periodically overhauled and revised, contrary to the opinion of those who would hold it as a sacred and unchangeable revelation from on high to the fathers, and Jefferson and later legal authorities have proposed that all laws should be enacted with a time limit, at the expiration of which they either are automatically repealed or must be reënacted. The provisions of the code of ethics of the American Veterinary Medical Association are listed and discussed below:

1. All members are expected to conduct themselves at all times as professional gentlemen.

In its spirit this provision will be concurred in quite generally. In its interpretation there will be a wide divergence of opinion. One may interpret this to mean that in one's private life, as well as in one's professional life, the Association may hold its members to a standard of conduct consistent with a statement of conventional conduct openly accepted by, though perhaps privately objectionable to, a vocal majority. Another will hold that one's professional conduct and one's private affairs are two different matters, and that one's private affairs are not the business of the profession or of the public at large. The word gentleman itself has fallen on hard times and has not the generally accepted meaning it once had in certain times and places; it hever had the same meaning at all times and in all places where English is spoken. Among other things it has implied a man who would give his seat in a street-car to a lady, but here again it has left a wide margin of interpretation as to what was a lady, and in a crowded New York subway train at the rush hour it would be difficult to separate ladies and gentlemen from others on such a basis. With out shifting standards of behavior the word gentle-

man has become a highly individual personal concept, and many men feel that there is more honor in being known as a man than as a gentleman.

2. No member shall assume an academic title or degree which has not been conferred upon him by an institution of learning in good standing.

There was probably a time when this provision had some point and usefulness. It is doubtful if it has much at present; we hope it hasn't. At a time when there are professors of acrobatics and doctors of charlatanry of all sorts, the misappropriation of titles and the assumption of cheaply obtained degrees would seem to be a waste of time and effort. Even the Ph.D.'s of great universities seem in many cases to have been too easily obtained and to mean little as regards ability and achievement. To violate this provision of the code would stamp a man as dumb, rather than unethical.

3. No member shall attempt to undermine or injure the professional standing of another by unfairly or unnecessarily criticizing his professional work.

Here is food for thought. In a world where all veterinarians were fair and intelligent persons, we could take our criticisms of our colleagues direct to them and discuss matters in a friendly way, and everyone would benefit from the proceeding. The world is not yet so generously supplied with fair and intelligent veterinarians. As a matter of fact we do criticize one another, mostly with professional colleagues and usually with an attempt to be fair. It is inconceivable that we should not do this, nor does the code forbid it. But the joker is this: Where is the border line between fair and necessary criticism and unfair and unnecessary criticism, and how do we determine whether the criticism of a colleague is or is not intended to undermine or injure his professional standing? Another big question is this: How far should one go in protecting an incompetent veterinarian—leaving out the question of his honesty—and to what extent is the public entitled to protection against incompetency? As long as the latter question is left to the veterinarian to answer, he can answer it to suit himself, but if the public ever raises the question it will answer it by a demand for protection.

4. In consultation cases the veterinarian in attendance must give the opinion of the consultant to the client in the presence of all three, or the consultant transmit it in writing to the client through

the attending veterinarian, and the consultant must not revisit the patient except by invitation from or by agreeemnt with the attending veterinarian.

This section, like Section 3, provides protection to professional colleagues, but it is not aimed at the provision of protection to clients. Consultants, whether veterinarians or physicians, habitually protect their colleagues. The consultant may find that the diagnosis was wrong and the treatment unsound, but his wording of the case to the client commonly begins with the confirmation of the diagnosis by the attending medical man and diplomatically modifies treatment. The ethical concept here does not seem to be as lofty as might be wished, and here, as in the practice of human medicine, one scents something that savors of professions as close corporations. A sound ethic would contemplate rather specifically the preservation of the rights of all parties concerned, including the veterinarians, the clients and the patients. What is omitted from this provision seems as significant as what is said.

5. In advertising, a veterinarian shall confine himself to his address and telephone number; may advertise that he has a properly equipped hospital, but not make it appear that he has unusual equipment unless he actually has it; shall not use large display advertisements, cuts or pictures; and must not advertise specific medicines or plans of treatment, or use posters, illustrated stationery, newspaper puffs, etc.

There is nothing in the code to indicate that certain sins are cardinal sins and others venial sins, so we must assume that this section ranks in importance with any other. However, the provisions of this section are quite habitually violated by members of the American Veterinary Medical Association and objections are brought to the attention of the Association only occasionally. There is always the likelihood, when regulations are violated with impunity, that there is relatively little sentiment in favor of enforcement, and the question may be raised as to whether this is the case here. If it is the case, the section should be repealed on the theory that dead things should be buried.

However, the writer is inclined to think that while some of the things mentioned as unethical may actually be so since they constitute a form of unfair competition, the matter as a whole is not one of ethics but actually of esthetics. Certainly a letter-head on which one-third of the space is filled with a spirited picture of a trotting horse has little advertising value, but it is an excellent

example of what most of us would regard as evidence of bad taste. General evidence of good taste on the part of veterinarians would be a great asset to the profession in attaining the status of a learned profession, and perhaps we should have a code of esthetics as well as a code of ethics.

6. No member shall prepare, advertise or sell any medicine, remedy or prophylactic of which he refuses to disclose the composition, nor propose to cure by secret medicine, nor guarantee cure.

It is obvious that the veterinarian who has any spiritual solidarity with his profession will endeavor to assist the profession in every way, and to that end will make public any discoveries which would advance the standing of the profession or enlarge our knowledge of veterinary science. It is perhaps easier for the man employed in state medicine to do this than for the practitioner or the commercial house, since the former is paid to do this thing and the latter make their discoveries at their own expense. However, it is true in the latter cases, as in the former case, that if a man wishes to retain professional standing, and not transfer to the status of a business man, he also will wish to advance veterinary science and to promote the welfare of the profession in all possible ways. As far as commercial houses are concerned, the veterinarian in practice should require from them detailed scientific evidence as to the value of their products on the ground that veterinary medicine is a science, not a faith, and as such requires scientific evidence for its ideas and its procedures. There is no doubt but what some veterinarians in practice keep to themselves useful findings on the ground that they discovered them and that there is no sense in giving them to their "competitors." As far as disciplinary action is concerned, probably little can be done or will be done about this. The remedy will lie in the development of a personnel from future veterinary students who appreciate that almost all they have of knowledge they owe to the living and dead who gave it to them, and who in return are glad to pay part of that debt by sharing their discoveries with others.

7. The last section of the code provides that charges against members for unethical conduct must be presented to the Executive Board.

Throughout the code of ethics there is the same striking omission that characterizes the Apostles' Creed. The code is mostly a statement of things unethical and forbidden; the Creed is mostly a statement of belief in things and events. Both of them

omit the positive ethical dicta incorporated in the Golden Rule and the Sermon on the Mount. The Creed was written in an age when faith was everything, but the church today is taking more cognizance of ethical conduct as the expression of the faith within. The code was written in modern times, but it leans too heavily towards the "Verboten!" It is too negative and lacks the stimulating value of positive and constructive statement. Perhaps it is time to write a new code or in some other way to keep before our veterinary students and the profession the positive and constructive ideas of honesty, integrity, courtesy, courage, tolerance, kindliness, thoughtfulness and good sportsmanship as the summation of ethics. If we have these virtues, and if we have good taste and good judgment, we can make a code of ethics as useless as policemen would be in a world of honest and intelligent persons. In fact the code savors too strongly of the police; it could be reworded to advantage in the language of the teacher and the minister. There is less value in the language of the police court than in these words of Paul: "Whatsoever things are true, whatsoever things are honorable, whatsoever things are just, whatsoever things are pure, whatsoever things are lovely, whatsoever things are of good report; if there be any virtue, and if there be any praise, think on these things."

12TH STREET VIADUCT

Three-deck viaduct connecting the Kansas City business district with the second largest stockyards in the world

ACCURACY OF THREE COOPERATING LABORATORIES IN DETECTING PULLORUM DISEASE BY THE AGGLUTINATION TEST*

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The widespread distribution and economic importance of pullorum disease has led to extensive application of the agglutination test, since adapted by Jones.[1] A review of publications [2-23] pertaining to the subject reveals a diversity of opinion, both regarding methods and accuracy of results from the use of the test. This is particularly true with respect to the constancy and accuracy of repeated tests and the uniformity of interpretation by different laboratories.

To obtain further information on these phases of the test and to establish standardization in technic, if possible, a coöperative arrangement was entered upon by three laboratories, each laboratory assuming equal responsibility in planning and conducting the work. These laboratories had done considerable routine testing previous to this time.[13,22,24] In this routine work a high degree of accuracy, as indicated by repeated tests and autopsy, had been obtained.

MATERIAL AND METHODS

The plan agreed upon called for each laboratory procuring thirty fowls, fifteen reactors to the agglutination test and fifteen non-reactors; to draw three blood samples from each fowl every four weeks for four times; each laboratory to receive blood samples from all fowls at every bleeding; to use key numbers for the samples and withhold these until the project was completed; to make a diagnosis for each blood sample at the completion of each series and send a report to the other laboratories. It was

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optional with each laboratory as to the number and method of tests to be applied to each sample.

Fifteen reacting and fifteen non-reacting fowls were procured by laboratories 1 and 3 before the first test. Laboratory 2 procured fourteen reactors and fifteen non-reactors before the first test and the fifteenth reactor before the second. The reactors were from commercial flocks in connection with which routine testing had been applied. The fowls and flocks from which they came were chosen at random and were not especially selected. The non-reactors were to be from pullorum-free flocks but this was not possible at that time at laboratories 1 and 3.

The fowls at laboratories 1 and 3 were all mature S. C. White Leghorn females. Those at laboratory 2 were fifteen mature negative-reacting White Wyandotte females and fifteen mature positive-reacting fowls of three breeds, including both sexes. The latter fifteen consisted of fourteen females: twelve R. I. Reds, one Light Sussex and one B. P. Rock, and one R. I. Red male.

Blood samples: Three blood samples were obtained from each fowl at each bleeding. This was done by nicking the ulnar vein and collecting about 1.5 cc of blood in 13x100-mm. serological test-tubes. The tubes were at once tightly corked and placed horizontally to clot. No disinfectants were used. One sample was kept at the laboratory where bled and the others expressed to the respective laboratories on the date bled. The samples contained the entire blood and were shipped in wooden boxes without ice; consequently some were hemolyzed, though on the whole they were satisfactory. The greatest distance between laboratories was 425 miles and samples shipped that distance were received approximately 24 to 36 hours and tested within 48 hours after bleeding.

Technic of the test: In each of the laboratories, antigen was prepared with four strains of *Salmonella pullora* which were obtained originally from the Veterinary Division of the Massachusetts Agricultural College, about 1923, and had been used for routine testing in each laboratory for several years.

Difco Bacto nutrient agar (1.5 per cent agar), to which 1.5 per cent peptone was added, was used by the three laboratories. Laboratory 1 also added 2.0 per cent gelatin. The cultures were incubated about 48 hours at approximately 37° C. in 25x200-mm. test-tubes.

TABLE I—*Testing technic*[1] *of laboratories 1, 2 and 3*

Laboratory	Method	Antigen[2]				Amount of Serum (cc)	Incubation					Read at		
		Density		pH	Amount (cc)		37° C.		Room Temperature		Hours in Refrigerator	Hours	Minutes	Minutes
		Gates cm.	McFarland				Hours	Minutes	Hours	Minutes				
1	Rapid	0.50 cm.	—	7.2 to 7.4[3]	0.05	0.005	—	—	—	5	—	—	2	5
	Tube	10.00 cm.	—	—	2.00 2.00 2.00 2.00	0.040 0.020 0.010 0.005	48	—	24	—	—	72	—	—
2	Rapid	—	50 x 0.75	7.8	0.02	0.040	—	5	—	2 to 3	—	—	5	7 to 8
	Tube	—	0.50	7.4[4]	2.00 2.00 2.00	0.800 0.400 0.020	24	—	24	—	—	48	—	—
3	Rapid	0.55 cm.	—	7.4	0.03	0.020	—	5	—	—	—	—	5	—
	Tube	See footnote 5	—	8.8[6]	2.00 2.00 2.00	0.040 0.020 0.010	24	—	—	—	24	48	—	—

[1]Remainder of information pertaining to test technic found in text.
[2]One cc of a 1% NaOH solution added to 100 cc of antigen used for the tube test by laboratories 2 and 3, and 1.0 cc of a 4 per cent NaOH solution to each 100. cc by laboratory 1.
[3]pH 7.2 for first three and pH 7.4 for fourth cooperative tests. Each represented average of three readings.
[4]pH before NaOH was added.
[5]Stock antigen standardized to density of 17.0 mm. on Gates nephelometer and this diluted fifteen times to standardize for use.
[6]pH after NaOH was added.

The stock antigen for the tube method was prepared by washing off the growth with 0.5 per cent phenolized physiological saline at the three laboratories. It was standardized just before use with physiological saline to which phenol was added as follows: laboratory 1, 0.5 per cent; laboratory 2, 0.1 per cent; laboratory 3, none. The rapid antigen was washed off with 12 per cent sodium chlorid in 0.25 per cent phenol in distilled water by laboratory 1, and 0.5 per cent by laboratories 2 and 3. The antigens were stored at approximately 7° C.

Further details in connection with the testing technic are included in table I.

Autopsy procedure and differential tests: In performing the autopsies the usual aseptic precautions were observed and examinations made of all viscera for gross lesions.

With very few exceptions, four or more ova were cultivated from the positive-reacting fowls. Cultures obtained were examined for morphological characteristics, and checked by Gram's stain. Acid and gas production in dextrose and mannite and absence in maltose and lactose was taken as an indication of *S. pullora*. In addition, laboratory 1 used a culture from each of its positive-reacting fowls for conducting agglutination tests with positive and negative sera.

In case of the negative fowls the ovaries were crushed and cultures were made from this material. Usually four or more cultures were made from the crushed ovary. Tissues showing gross lesions of any nature also were cultured and in addition the liver in the majority of the negative reactors.

Agglutination Tests and Diagnoses

Serological, gross and bacteriological diagnoses are included in tables II, III and IV. Table V includes all tests conducted by laboratory 1 on laboratory 1 samples at the first coöperative test. Table VI includes all tests conducted by laboratory 2 on laboratory 3 samples at the third coöperative test. Table VII includes all tests conducted by laboratory 3 on laboratory 3 samples at the second coöperative test. The agglutination tests of these samples were selected as representative of the various series of tests, consequently the remainder are omitted. Similar series of tests were used by the respective laboratories in establishing all the diagnoses.

TABLE II—*Agglutination and postmortem diagnoses of fowls in laboratory 1.*

Fowl[1]	Routine Test Diagnoses[2]	Cooperative Tests[3]												Postmortem Findings	
		First			Second			Third			Fourth			Macroscopic	Bacteriological
		Sample	Laboratory 1	2	3	Sample	1	2	3	Sample	1	2	3	Sample	1	2	3		
B64471	P	19	P	P	P	16	P	P	P	4	P	P	P	17	P	P	P	32 typical ova	*S. pullora*
B64370	P	29	P	P	P	11	P	P	P	16	P	P	P	13	P	P	P	39 typical ova	*S. pullora*
B64577	P	20	P	P	P	12	P	P	P	24	P	P	P	3	P	P	P	4 typical ova	*S. pullora*
B64469	P	21	P	P	P	22	P	P	P	27	P	P	P	19	P	P	P	21 typical ova	*S. pullora*
B64516	P	24	P	P	P	28	P	P	P	14	P	P	P	12	P	P	P	14 typical ova	*S. pullora*
B64519	P	23	P	P	P	27	P	P	P	1	P	P	P	22	P	P	P	34 typical ova	*S. pullora*
B64558	P	13	P	P	P	7	P	P	P	6	P	P	P	14	P	P	P	21 typical ova	*S. pullora*
B64495	P	16	P	P	P	13	P	P	P	7	P	P	P	8	P	P	P	19 typical ova	*S. pullora*
B64661	P	15	P	P	P	1	P	P	P	11	P	P	P	26	P	P	P	10 typical ova	*S. pullora*
B64642	P	17	P	P	P	4	P	P	P	28	P	P	P	27	P	P	P	23 typical ova	*S. pullora*
B64314	P	18	P	S	P	19	P	P	P	2	P	P	P	4	P	P	P	4 typical ova	*S. pullora*
B64477	P	25	P	P	P	8	P	P	P	8	P	P	P	18	P	P	P	20 typical ova	*S. pullora*
B64367	P	28	P	P	P	23	P	P	P	15	P	P	P	21	P	P	P	2 typical ova	*S. pullora*
B64308	P	30	P	P	P	2	P	P	P	23	P	P	P	7	P	P	P	85 typical ova	*S. pullora*
B64515	P	27	P	P	P	6	P	S	P	19	P	P	P	5	P	S	P	11 typical ova	*S. pullora*
A35051	N	11	N	N	N	10	N	N	N	13	N	N	N	15	N	N	N	No lesions	Negative
A35065	N	22	N	N	N	26	N	N	N	25	N	N	N	2	N	N	N	No lesions	Negative
AM2078[4]	N	9	S	N	S	—	—	—	—	—	—	—	—	—	—	—	—	No lesions	Negative
AM2082	N	1	N	N	N	15	N	N	N	22	N	N	N	10	N	N	N	No lesions	Negative
A35045	N	6	N	N	N	9	N	N	N	18	N	N	N	1	N	N	N	No lesions	Negative
A35061	N	12	N	N	N	24	N	N	N	12	N	N	N	24	N	N	N	No lesions	Negative
A35033	N	3	N	N	N	3	N	N	N	20	P	P	P	23	P	P	P	1 typical ovum	*S. pullora*
A35066	N	2	N	N	N	18	N	N	N	26	N	N	N	6	N	N	N	No lesions	Negative
A35049	N	4	N	N	N	17	N	N	N	9	N	N	N	16	N	N	N	No lesions	Negative

TABLE II—*Agglutination and postmortem diagnoses of fowls in laboratory 1—Continued*

Fowl[1]	Routine Test Diagnoses[2]	Cooperative Tests[3]												Postmortem Findings	
		First			Second			Third			Fourth			Macroscopic	Bacteriological
		Sample	Laboratory 1	2	3	Sample	Laboratory 1	2	3	Sample	Laboratory 1	2	3	Sample	Laboratory 1	2	3		
A35057	N	8	N	N	N	25	N	N	N	3	N	N	N	20	N	N	N	No lesions	Negative
A35075	N	7	N	N	N	21	N	N	N	5	N	N	N	25	N	N	N	No lesions	Negative
A35044	N	26	N	N	N	20	N	N	N	17	N	N	N	9	N	N	N	No lesions	Negative
A35067	N	5	N	N	N	5	N	N	N	10	N	N	N	11	N	N	N	No lesions	Negative
A35035	N	10	N	N	N	14	N	N	N	21	N	N	N	28	N	N	N	No lesions	Negative
A35054[5]	N	14	N	N	N	—	—	—	—	—	—	—	—	—	—	—	—	No lesions	Negative

[1]All mature females. S. C. White Leghorns.
[2]Diagnoses made at time of conducting routine field test, Sept. 25, 1929, for positive reactors, and Nov. 19, 1929, for negative reactors.
[3]First, second, third and fourth cooperative tests conducted Feb. 12, Mar. 12, Apr. 9 and May 6, 1930 respectively.
[4]Died Feb. 16, 1930.
[5]Died Feb. 21, 1930.
N = Negative to agglutination test.
P = Positive to agglutination test.
S = Suspicious to agglutination test.

TABLE III—*Agglutination and postmortem diagnoses of fowls in laboratory 2.*

Fowl[1]	Routine Test Diagnoses[2]	Cooperative Tests[3]												Postmortem Findings	
		First			Second			Third			Fourth			Macroscopic	Bacteriological
		Sample	Laboratory 1	2	3	Sample	1	2	3	Sample	1	2	3	Sample	1	2	3		
1	P	1	P	P	P	959	P	P	P	1	P	P	P	1	P	P	P	Typical ova	*S. pullora*
2	P	2	P	P	P	023	P	P	P	2	P	P	P	4	P	P	P	Typical ova	*S. pullora*
3	P	3	P	P	P	085	P	P	P	3	P	P	P	6	P	P	P	Tpyical ova	*S. pullora*
4	P	4	P	P	P	012	P	P	P	4	P	P	P	8	P	P	P	Typical ova	*S. pullora*
5	P	5	P	P	P	986	P	P	P	5	P	P	P	15	P	P	P	Typical ova	*S. pullora*
6	P	6	P	P	P	328	P	P	P	6	P	P	P	12	P	P	P	Typical ova	*S. pullora*
7	P	7	P	P	P	982	P	P	P	7	P	P	P	14	P	P	P	Typical ova	*S. pullora*
8	P	8	P	P	P	971	P	P	P	8	P	P	P	16	P	P	P	Typical ova	*S. pullora*
9	P	9	P	P	P	958	P	P	P	9	P	P	P	18	P	P	P	Typical ova	*S. pullora*
10	P	10	P	P	P	272	P	P	P	10	P	P	P	10	P	P	P	Typical ova	*S. pullora*
11	P	11	P	P	P	38	P	P	P	11	P	P	P	33	P	P	P	Small abnormal ova	Contamination
12	P	12	P	P	P	024	P	P	P	12	P	P	P	24	P	P	P	Cystic ovary	*S. pullora*
13	P	13	P	P	P	980	P	P	P	13	P	P	P	130	P	P	P	Pericarditis	Negative
14	P	14	P	P	P	14B	P	P	P	14	P	P	P	28	P	P	P	Typical ova	*S. pullora*
182	P					182	P	P	P	182	P	P	P	0	P	P	P	Typical ova	*S. pullora*
15	N	15	N	N	N	13	N	N	N	15	N	N	N	11	N	N	N	No lesions	Negative
16	N	16	N	N	N	18	N	N	N	16	N	N	N	9	N	N	N	No lesions	Negative
17	N	17	N	N	N	15	N	N	N	17	N	N	N	19	N	N	N	No lesions	Negative
18	N	18	N	N	S	20	N	N	S	18	N	N	N	5	N	N	S	No lesions	Negative
19	N	19	N	N	N	17	N	N	N	19	N	N	N	2	N	N	N	No lesions	Negative
20	N	20	N	N	N	22	N	N	N	20	N	N	N	23	N	N	N	No lesions	Negative
21	N	21	N	N	N	19	N	N	N	21	N	N	N	21	N	N	N	No lesions	Negative
22	N	22	N	N	N	24	N	N	N	22	N	N	N	13	N	N	N	No lesions	Negative

TABLE III—*Agglutination and postmortem diagnoses of fowls in laboratory 2—Continued*

Fowl[1]	Routine Test Diagnoses[2]	Cooperative Tests[3]												Postmortem Findings	
		First			Second			Third			Fourth			Macroscopic	Bacteriological
		Sample	Laboratory 1	2	3	Sample	1	2	3	Sample	1	2	3	Sample	1	2	3		
23	N	23	N	N	N	21	N	N	N	23	N	N	N	17	N	N	N	No lesions	Negative
24	N	24	N	N	N	26	N	N	N	24	N	N	N	7	N	N	N	No lesions	Negative
25	N	25	N	N	N	23	N	N	N	25	N	N	N	25	N	N	N	No lesions	Negative
26	N	26	N	N	N	28	N	N	N	26	N	N	N	26	N	N	N	No lesions	Negative
27	N	27	N	N	N	25	N	N	N	27	N	N	N	40	N	N	N	No lesions	Negative
28	N	28	N	N	N	30	N	N	N	28	N	N	N	3	N	N	N	No lesions	Negative
29	N	29	N	N	N	27	N	N	N	29	N	N	N	29	N	N	N	No lesions	Negative

[1]Fowl 13 is a mature male; all others mature females. Heavy breeds.
[2]Diagnoses made at time of conducting routine field test—Dec. 18, 1929, for positives and Nov. 12, 1929, for negatives.
[3]First, second, third and fourth cooperative tests conducted Feb. 12, Mar. 12, Apr. 9 and May 6, 1930, respectively.
N = Negative to agglutination test.
P = Positive to agglutination test.
S = Suspicious to agglutination test.

TABLE IV—*Agglutination and postmortem diagnoses of fowls in laboratory 3.*

Fowl[1]	Routine Test Diagnoses[2]	Cooperative Tests[3]												Postmortem Findings	
		First			Second			Third			Fourth			Macroscopic	Bacteriological
		Sample	Laboratory 1	2	3	Sample	1	2	3	Sample	1	2	3	Sample	1	2	3		
281-8	P	281-8	P	P	P	17	P	P	P	22	P	P	P	3	P	P	P	3 typical ova	*S. pullora*
414	P	414	P	P	P	6	P	P	P	6	P	P	P	11	P	P	P	7 typical ova	*S. pullora*
434	P	434	P	P	P	7	P	P	P	26	P	P	P	15	P	P	P	20 typical ova	*S. pullora*
437	P	437	P	P	P	19	P	P	P	23	P	P	P	6	P	P	P	8 typical ova	*S. pullora*
456[4]	P	456	P	P	P	14	P	P	P	—	—	—	—	—	—	—	—	12 typical ova	*S. pullora*
475	P	475	P	P	P	8	P	P	P	10	P	P	P	16	P	P	P	3 typical ova	*S. pullora*
536[5]	P	536	P	P	P	15	P	P	P	5	P	P	P	—	—	—	—	24 typical ova	*S. pullora*
546	P	546	P	P	P	2	P	P	P	2	P	P	P	8	P	P	P	4 typical ova	*S. pullora*
558	P	558	P	P	P	3	P	P	P	24	P	P	P	22	P	P	P	4 typical ova	*S. pullora*
564	P	564	P	P	P	11	P	P	P	19	P	P	P	5	P	P	P	25 typical ova	*S. pullora*
569	P	569	P	P	P	23	P	P	P	20	P	P	P	12	P	P	P	5 typical ova	*S. pullora*
576	P	576	P	P	P	20	P	P	P	17	P	P	P	1	P	P	P	9 typical ova	*S. pullora*
742-8	P	742-8	P	P	P	29	P	P	P	14	P	P	P	24	P	P	P	5 typical ova	*S. pullora*
969	P	969	P	P	P	25	P	P	P	27	P	P	P	18	P	P	P	7 typical ova	*S. pullora*
1559-6	P	1559-6	P	P	P	4	P	P	P	12	P	P	P	26	P	P	P	2 typical ova	*S. pullora*
125-8[6]	N	125-8	N	N	N	24	N	N	N	4	N	N	N	—	—	—	—	No lesions	Negative
307-8	N	307-8	N	N	N	27	N	N	N	11	N	N	N	25	N	N	N	No lesions	Negative
387-8	N	387-8	N	N	N	18	N	N	N	8	N	N	N	17	N	N	N	No lesions	Negative
464-7	N	464-7	N	N	N	22	N	N	N	28	N	N	N	20	N	N	N	No lesions	Negative
505-8	N	505-8	N	N	N	16	N	N	N	7	N	N	N	9	N	N	N	No lesions	Negative
616-7	N	616-7	N	N	N	1	N	N	N	21	N	N	N	14	N	N	N	No lesions	Negative
647-8	N	647-8	N	N	N	26	N	N	N	9	N	N	N	19	N	N	N	No lesions	Negative
705-8	N	705-8	N	N	N	10	N	N	N	1	N	N	N	21	N	N	N	No lesions	Negative
720-8	N	720-8	N	N	N	13	N	N	N	15	N	N	N	23	N	N	N	No lesions	Negative

TABLE IV—*Agglutination and postmortem diagnoses of fowl in laboratory 3—Continued*

Fowl[1]	Routine Test Diagnoses[2]	Cooperative Tests[3]										Postmortem Findings	
		First			Second			Third			Fourth			Macroscopic	Bacteriological
		Sample	Laboratory 1	2	3	Sample	Laboratory 1	2	3	Sample	Laboratory 1	2	3		
726-8	N	726-8	N	N	N	9	N	N	N	13	N	N	N	7	N	N	N	No lesions	Negative
783-8	N	783-8	N	N	N	30	N	N	N	25	N	N	N	4	N	N	N	No lesions	Negative
968-8	N	968-8	N	N	N	21	N	N	N	18	N	N	N	10	N	N	N	No lesions	Negative
976-8	N	976-8	N	N	N	12	N	N	N	16	N	N	N	2	N	N	N	No lesions	Negative
1116-8[7]	N	1116-8	N	N	N	28	N	N	N	—	—	—	—	—	—	—	—	No lesions	Negative
1337-7	N	1337-7	N	N	N	5	N	N	N	3	N	N	N	13	N	N	N	No lesions	Negative

[1]All mature females. S. C. White Leghorns.
[2]Diagnoses made at time of conducting routine field tests—Oct. 3 and Nov. 23, 1929, and Jan. 17, 1930, for positives and Jan. 4, 1930, for negatives.
[3]First, second, third and fourth cooperative tests conducted Feb. 12, Mar. 12, Apr. 9 and May 6, 1930, respectively.
[4]Died Apr. 1, 1930.
[5]Died Apr. 17, 1930.
[6]Died Apr. 7, 1930.
[7]Died Mar. 10, 1930.
N = Negative to agglutination test.
P = Positive to agglutination test.
S = Suspicious to agglutination test.

TABLE V—*Representative series of agglutination tests conducted by laboratory 1—all tests of laboratory 1 samples at first cooperative test.*

	Method

TABLE V.—*Representative series of agglutination tests conducted by laboratory I—all tests of laboratory I samples at first cooperative test.*

Fowl	Sample	Method										Diagnoses
		Rapid						Tube				
		First Test		Second Test		Third Test		1:50 Dilution	1:100 Dilution	1:200 Dilution	1:400 Dilution	
		First[1] Reading	Second[2] Reading	First Reading	Second Reading	First Reading	Second Reading					
AM2082	1	—	—	—	—	—	—	—	—	—	—	N
A35066	2	—	—	—	—	—	—	—	—	—	—	N
A35033	3	—	—	—	—	—	—	—	—	—	—	N
A35049	4	—	—	—	—	—	—	—	—	—	—	N
A35067	5	—	—	—	—	—	—	—	—	—	—	N
A35045	6	—	—	—	—	—	—	—	—	—	—	N
A35075	7	—	—	—	—	—	—	—	—	—	—	N
A35057	8	—	—	—	—	—	—	—	—	—	—	N
AM2078	9	—	—	—	—	—	—	+ +	+ +	+ ?	—	S
A35035	10	—	—	—	—	—	—	—	—	—	—	N
A35051	11	—	—	—	—	—	—	—	—	—	—	N
A35061	12	—	—	—	—	—	—	—	—	—	—	N
B64558	13	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	P
A35054	14	—	—	—	—	—	—	—	—	—	—	N
B64661	15	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	P
B64495	16	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	P
B64642	17	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	P
B64314	18	+	+	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	P
B64471	19	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	P
B64577	20	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	+ ?	P
B64469	21	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	+	P
A35065	22	—	—	—	—	—	—	—	—	—	—	N
B64519	23	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	P
B64516	24	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	P
B64477	25	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	P
A35044	26	—	—	—	—	—	—	—	—	—	—	N
B64515	27	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	P
B64367	28	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	P
B64370	29	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	P
B64308	30	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	P

[1]Reading made two minutes after dilution.
[2]Reading made five minutes after dilution.
— = Negative reaction.
+ = Very slight agglutination but regarded as negative reaction.
+ + = Slight agglutination regarded as suspicious reaction.
+ + + = Complete agglutination or nearly so.

Discussion

All three laboratories conducted several tests in connection with each blood sample in the coöperative testing. The customary procedure in this regard is shown in tables V, VI and VII. Had each laboratory applied only its respective routine test the agglutination test diagnoses would have been in complete agreement with the autopsies, with the exception of fowl A35033 in laboratory 1, as noted later. Autopsy findings in this fowl were in complete accord with the diagnoses of the third and fourth coöperative tests.

Table VI—*Representative series of agglutination tests conducted by laboratory 2—all tests of laboratory 3 samples at third cooperative test.*

Fowl	Sample	Method					Diagnoses
		Rapid		Tube			
		First[1] Reading	Second[2] Reading	1:25 Dilution	1:50 Dilution	1:100 Dilution	
705-8	1	−	−	−	−	−	N
546	2	+ + +	+ + +	+ + +	+ + +	+ + +	P
1337-7	3	−	−	+	−	−	N
125-8	4	−	−	−	−	−	N
536	5	+ + +	+ + +	0	+ + +	+ + +	P
414	6	+ + +	+ + +	0	+ + +	+ + +	P
505-8	7	−	−	−	−	−	N
387-8	8	−	−	−	−	−	N
647-8	9	−	−	−	−	−	N
475	10	+ + +	+ + +	0	+ + +	+ + +	P
307-8	11	−	−	−	−	−	N
1559-6	12	+ + +	+ + +	+ + +	+ + +	+ + +	P
726-8	13	−	−	−	−	−	N
742-8	14	+ + +	+ + +	H	+	+ + +	P
720-8	15	−	−	−	−	−	N
976-8	16	−	−	−	−	−	N
576	17	+ + +	+ + +	+ + +	+ + +	+ + +	P
968-8	18	−	−	−	−	−	N
564	19	+ + +	+ + +	+ + +	+ +	+ + +	P
569	20	+ + +	+ + +	+ +	+ +	+ +	P
616-7	21	−	−	−	−	−	N
281-8	22	+ + +	+ + +	0	+ + +	+ + +	P
437	23	+ + +	+ + +	+ + +	+ + +	+ + +	P
558	24	+ + +	+ + +	+ + +	+ + +	+ +	P
783-8	25	−	−	−	−	−	N
434	26	+ + +	+ + +	+ +	+ +	+ + +	P
969	27	+ + +	+ + +	+	+ + +	+ + +	P
464-7	28	−	−	−	−	−	N

[1]Reading made five minutes after dilution.
[2]Reading made eight minutes after dilution.
− = Negative reaction.
+ = Negative reaction.
+ + = Positive reaction.
+ + + = Positive reaction.
0 = No serum.
H = Hemolyzed and impossible to read.

The data show that a total of 1,041 agglutination test diagnoses (table VIII) were made during the four coöperative tests, of which 531 were positive, 8 suspicious and 502 negative. The only disagreements in the test diagnoses were in the eight classed as suspicious and involving fowls B64314, B64515, AM2078 (table II) and fowl 18 (table III), constituting 0.77 per cent of the 1,041 diagnoses. In no case did a laboratory make a positive test diagnosis when another made a negative diagnosis.

Autopsy of the 46 fowls which proved positive to the agglutination test by the three laboratories definitely established *S. pullora* in 44 of them (95.65 per cent). *S. pullora* was not isolated from female 12 and male 13 (table III). All 46 positive fowls showed typical lesions of pullorum disease, thus indicating 100

TABLE VII—*Representative series of agglutination tests conducted by laboratory 3 —all tests of laboratory 3 samples at second cooperative test*

Fowl	Sample	Method				Diagnoses
		Rapid Final Reading[1]	Tube 1:50 Dilution	Tube 1:100 Dilution	Tube 1:200 Dilution	
616-7	1	−	−	−	−	N
546	2	+ + +	+ + +	+ + +	+ + +	P
558	3	+ + +	+ + +	+ + +	+ + +	P
1559-6	4	+ + +	+ + +	+ + +	+ + +	P
1337-7	5	−	−?	−	−	N
414	6	+ + +	+ + +	+ + +	+ + +	P
434	7	+ + +	+ + +	+ + +	+ + +	P
475	8	+ + +	+ + +	+ + +	+ + +	P
726-8	9	−	−	−	−	N
705-8	10	−	−	−	−	N
564	11	+ + +	+ + +	+ + +	+ + +	P
976-8	12	−	−	−	−	N
720-8	13	−	−	−	−	N
456	14	+ + +	+ + +	+ + +	+ + +	P
536	15	+ + +	+ + +	+ + +	+ + +	P
505-8	16	−	−	−	−	N
281-8	17	+ + +	+ + +	+ + +	+ + +	P
387-8	18	−?	−?	−	−	N
437	19	+ + +	+ + +	+ + +	+ + +	P
576	20	+ + +	+ + +	+ + +	+ + +	P
968-8	21	−	−	−	−	N
464-7	22	−	−	−	−	N
569	23	+ + +	+ + +	+ + +	+ + +	P
125-8	24	−	−	−	−	N
969	25	+ + +	+ + +	+ + +	+ + +	P
647-8	26	−	−	−	−	N
307-8	27	−	−	−	−	N
1116-8	28	−	−	−	−	N
742-8	29	+ + +	+ + +	+ + +	+ + +	P
783-8	30	−	−	−	−	N

[1]Reading made five minutes after dilution.
− = Negative reaction.
+ + + = Complete agglutination or nearly so.

TABLE VIII—*Summary of agglutination test diagnoses of fowls in laboratories 1, 2 and 3*

Flock[1]	Laboratory Conducting Tests	Routine Test[2]				Cooperative Tests															
			Diagnosis			Fowls Tested				Classfication of Diagnoses											
										Positive				Suspicious				Negative			
		Fowls Selected	Positive	Suspicious	Negative	First	Second	Third	Fourth	First	Second	Third	Fourth	First	Second	Third	Fourth	First	Second	Third	Fourth
1	1	30	15	0	15	30	28	28	28	15	15	16	16	1	0	0	0	14	13	12	12
1	2					30	28	28	28	14	14	16	15	1	1	0	1	15	13	12	12
1	3					30	28	28	28	15	15	16	16	1	0	0	0	14	13	12	12
2	1					29	30	30	30	14	15	15	15	0	0	0	0	15	15	15	15
2	2	30	15	0	15	29	30	30	30	14	15	15	15	0	0	0	0	15	15	15	15
2	3					29	30	30	30	14	15	15	15	1	1	0	1	14	14	15	14
3	1					30	30	28	26	15	15	14	13	0	0	0	0	15	15	14	13
3	2					30	30	28	26	15	15	14	13	0	0	0	0	15	15	14	13
3	3	30	15	0	15	30	30	28	26	15	15	14	13	0	0	0	0	15	15	14	13
Totals		90	45	0	45	89	88	86	84	531				8				502			
Grand total tests										1041											

[1]Flocks 1, 2 and 3 were at laboratories 1, 2 and 3 respectively.
[2]Refers to routine field tests which provided the basis for obtaining the cooperative test fowls.

FIG. 1. Ovaries from 13 positive-reacting females of laboratory 2. (One ovary was destroyed before photograph was made.

per cent *S. pullora* infection. Autopsy of the 44 fowls which were not positive to the agglutination test failed to produce any evidence of infection.

The intervals (tables II, III and IV footnotes) between the date of the routine test and that of the fourth coöperative test were as follows: laboratory 1, 223 days for positives and 168 days for negatives; laboratory 2, 140 days for positives and 185 days for negatives; laboratory 3, 108 to 214 days for positives and 122 days for negatives. Notwithstanding the length of time during which the tests were applied and the number involved, these tables show that during the intervals between the routine

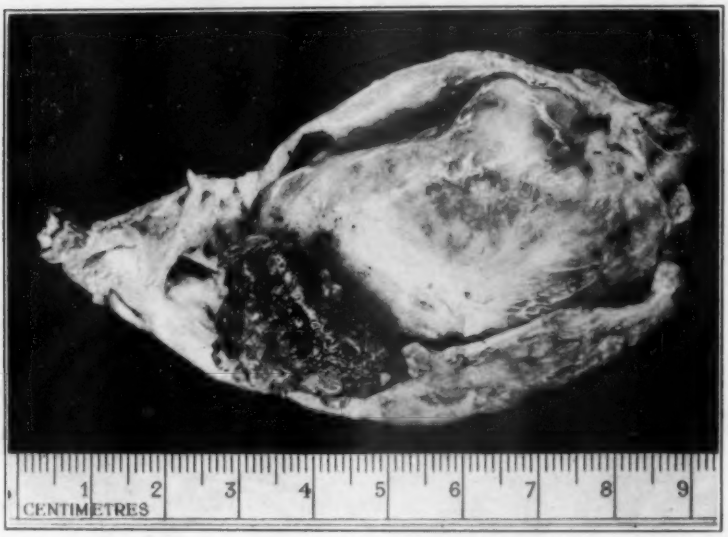

FIG. 2. Pericarditis of fowl 13 (male) of laboratory 2.

test which located the fowls and the last coöperative test, the only complete reversal in reaction was in the case of fowl A35033 (table II), which changed to positive in the third coöperative test. All test diagnoses of this fowl by each laboratory were in absolute agreement at each coöperative test. None of the fowls positive to the routine test ever gave a negative reaction.

In view of the nearly 100 per cent agreement in diagnosis, it is apparent that absolute standardization of the test was not essential to obtain a high degree of accuracy.

That the percentage of agreement in test diagnoses among the three laboratories and the constancy in reaction of the individual

fowl are higher than could be obtained in testing a large number of fowls is indicated by experimental and routine testing at these laboratories and elsewhere. In conducting many thousands of tests, sometimes fowls are encountered in which the blood sera show fluctuations in agglutinability of a nature not encountered in this coöperative test. Obviously such cases would increase somewhat the possibility of variable interpretations of the test.

Summary

1. Fifteen negative- and fifteen positive-reacting fowls were procured at random by each of the three laboratories and all surviving were bled four times at four-week intervals for coöperative agglutination testing for pullorum disease.

2. Three blood samples were drawn from each fowl at each bleeding and tested at British Columbia, Washington and Oregon laboratories, each applying its own technic.

3. A total of 1041 blood samples was tested and a diagnosis made for each—531 positives, 8 suspicious and 502 negatives, or a disagreement of 0.77 per cent of the 1041 diagnoses. The disagreements in agglutination test diagnoses among the laboratories were from negative or positive to suspicious and not negative to positive or the reverse.

4. One fowl changed from negative to positive, but none changed from positive to negative.

5. Postmortem findings of the 46 positive-reacting fowls definitely established *S. pullora* infection in 44 (95.65 per cent) and characteristic lesions in the other two indicated a probable 100 per cent *S. pullora* infection.

6. Postmortem findings of the 44 fowls which never gave a positive reaction were 100 per cent negative, as determined bacteriologically and by absence of *S. pullora* lesions.

7. All three laboratories were in absolute accord in the 342 test diagnoses of the fifteen positive- and fifteen negative-reacting fowls of laboratory 3, and postmortem examinations established the positive fowls infected and the negative fowls not infected, as determined by gross and bacteriological examinations.

References

[1]Jones, F. S.: The value of the macroscopic agglutination test in detecting fowls that are harboring *Bacterium pullorum*. Jour. Med. Res., xxvii (1913), n. s. 22 (4), pp. 481-495.

[2]Hooker, W. A.: Bibliography of bacillary white diarrhea infection of the fowl (1889-1927). Office of Exp. Sta., U. S. Dept. Agr. (1927).

[3]Mallmann, W. L.: An improved antigen for the agglutination test in bacillary white diarrhea. Jour. A. V. M. A., lxxi (1927), n. s. 24 (5), pp. 600-606.

[4]Fitch, C. P., and Lubbehusen, R. E.: The agglutination test as applied in the control of bacillary white diarrhea. Corn. Vet., xviii (1928), 1, pp. 19-27.

[6]Newsom, I. E., Cross, F., and Ufford, O. C.: On the accuracy of the agglutination test for *Bacterium pullorum* infection as shown by repeated tests on the same birds. Jour. A. V. M. A., lxxii (1928), n. s. 25 (5), pp. 611-617.
[6]Hinshaw, W. R., and Sanders, E. F.: Control of *Salmonella pullorum* infection. Mass. Agr. Exp. Sta. Control Series Bul. 43 (1928).
[7]Dalling, T., Mason, J. H., and Gordon, W. S.: Bacillary white diarrhea of chicks (B. W. D.). Vet. Jour., lxxxiii (1928), 11, pp. 555-565.
[8]Edwards, P. R., and Hull, F. E.: The accuracy of the agglutination test in the diagnosis of bacillary white diarrhea. Jour. A. V. M. A., lxxiii (1928), n. s. 26 (7), pp. 839-843.
[9]Tittsler, R. P., Heywang, B. W., and Charles, T. B.: The occurrence and significance of *Salmonella pullorum* in eggs. Pa. Agr. Exp. Sta. Bul. 235 (1928).
[10]Bushnell, L. D., and Brandly, C. A.: Some experiments on the control of bacillary white diarrhea. Jour. A. V. M. A., lxxiv (1929), n. s. 27 (4), pp. 444-453.
[11]Runnells, R. A.: Bacillary white diarrhea. Va. Agr. Exp. Sta. Bul. 265 (1929).
[12]Rettger, L. F.: The need of accepted scientific standards and rigid adherence to them, in pullorum disease control. Jour. A. V. M. A., lxxiv (1929), n. s. 27 (4), pp. 453-461.
[13]Biely, J.: Control of bacillary white diarrhea infection of poultry in British Columbia. Sci. Agr., ix (1929), 7, pp. 413-422.
[14]Kernkamp, H. C. H.: The results of repeated testing by the agglutination method for the detection of bacillary white diarrhea in adult chickens. Corn. Vet., xix (1929), 4, pp. 357-369.
[15]Bunyea, H., Hall, W. J., and Dorset, M.: A simplified agglutination test for pullorum disease. Jour. A. V. M. A., lxxv (1929), n. s. 28 (4), pp. 408-410.
[16]Bushnell, L. D., and Brandly, C. A.: Comparison of tube and slide agglutination tests for bacillary white diarrhea. Jour. A. V. M. A., lxxiii (1928), n. s. 26 (7), pp. 844-847.
[17]Edwards, P. R., and Hull, F. E.: The slide agglutination test in the detection of bacillary white diarrhea. Jour. A. V. M. A., lxxv (1929), n. s. 28 (5), pp. 626-628.
[18]Michael, S. T., and Beach, J. R.: An experimental study of tests for the detection of carriers of *Bacterium pullorum*. Hilgardia, iv (1929), 8, pp. 185-200.
[19]Edwards, P. R., and Hull, F. E.: The constancy of the agglutination test in the detection of bacillary white diarrhea. Jour. A. V. M. A., lxxv (1929), n. s. 28 (6), pp. 765-768.
[20]Anonymous: Conference of laboratory workers in pullorum disease (B. W. D.) eradication. News Release (1929).
[21]Rettger, L. F., McAlpine, J. G., and Warner, D. E.: A comparative study of the routine macroscopic agglutination and the intracutaneous (wattle) tests for *Bacterium pullorum* infection in poultry breeding stock. Jour. A. V. M. A., lxxvii (1930), n. s. 30 (1), pp. 47-57.
[22]Sawyer, C. E., and Hamilton, C. M.: Pullorum disease. West. Wash. Exp. Sta. Bul. 17-W, n. s. (1930).
[23]Dalling, T., and Warrack, G. H.: Bacillary white diarrhea (B. W. D.). Proc. Fourth World's Poultry Cong. (1930), pp. 420-426.
[24]Oregon Agricultural Experiment Station: Unpublished data.

Rabies on the Increase

Rabies shows some indications of being on the increase again, in certain localities, after a period of comparative freedom from the disease. Counties in widely separated parts of Michigan are now under quarantine and in certain sections of Illinois similar restrictions have recently become necessary. Five townships in Madison County (Ill.) have been quarantined, and recently it was necessary for eight persons in the vicinity of Edwardsville to submit to the Pasteur treatment, following bites inflicted by rabid animals. It is also reported that eighteen dogs, seven cows, two horses and two mules were afflicted with the disease in the vicinity of Edwardsville, and had to be killed. Several counties in Michigan have been quarantined for some time, and recently Lenawee and Osceola counties were added to the list. These counties will be under quarantine until August.

Doctor Dimock Called to Europe

Dr. W. W. Dimock, of the University of Kentucky, has received an invitation from John Crawford, M.R.C.V.S., of the British Bloodstock Agency, Ltd., to visit England and France for the purpose of consulting with veterinarians there on breeding problems in mares. Dr. Dimock planned to sail the latter part of June.

THE BLOOD PRESSURE OF THE PIG AND THE INFLUENCE OF NON-NERVOUS AND NERVOUS FACTORS ON THE CARDIOVASCULAR APPARATUS*

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I. Introduction

In this paper we present the results of studies in the pig on the normal blood pressure and on non-nervous and nervous influences on the cardiovascular mechanism. In later papers results will be presented showing the influence of drugs on the cardiovascular apparatus and the influence of nervous factors on respiration.

The studies reported in these papers are not claimed to be exhaustive. They were undertaken rather as a general survey of a broad field in the hope that they might indicate lines along which more detailed work in the pig might profitably proceed.

As far as we know, the literature contains no record of work in the pig similar to that reported in these communications. While most of the points covered have been studied by other workers in other species, we submit that the pig's economic importance demands that these and similar problems be investigated on the pig itself. A facile assumption that the conditions are the same as in some small animal is unscientific and not conducive to progress. A special physiology of farm animals must be developed. Many of the fathers of modern physiology studied function in the large animals. Later, for good reasons, the small animals received most of the attention of the mammalian physiologists and doubtless will continue to do so. Nevertheless, recent years have seen a revival of research work in the physiology of farm animals, and it is a good sign. We may therefore confidently look forward to the time, as yet in the distant future, when there shall have evolved a special physiology of these animals, such as must have been contemplated by Colin, and Chauveau, and R. M. Smith, and Ellenberger, and F. Smith and others.

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LITERATURE

As stated above, the literature known to us offers nothing for the pig on the problems covered in this paper. However, for those who wish to learn what has been done in other species, entry into this most extensive literature may be found through many writings, only a few of which can be cited here.[1, 2, 3, 10, 11, 13, 14, 15, 16, 17]

DESCRIPTION OF ANIMALS

The distribution by breed of the pigs is as follows: Tamworth, 17; Duroc Jersey, 4; Poland China, 3; not recorded, 2. By sex: castrated male, 17; uncastrated male, 2; female, 3; not recorded, 2.

The average weight of the pigs was 35 kg. (77 pounds). The lightest pig weighed 19 kg., the heaviest 57 kg. The weights of two-thirds of the animals fell between 30 and 40 kg., inclusive. The pigs ranged in age from about seven to ten months.

Practically all of the pigs were undersized, though most of them were in good condition. A few were mangy and two or three showed, on autopsy, chronic pleurisy and chronic pericarditis. However, no pig was suffering from acute disease, and we do not believe that any one was sufficiently unhealthy to have modified the experimental results significantly.

ANESTHESIA

In the early experiments urethane anesthesia alone was used. It soon appeared that the low blood pressures encountered were due to urethane, and in order to determine the true blood pressure, many of our subsequent experiments were begun under local anesthesia (usually procaine, 2 per cent; once apothesine, 2 per cent). Following this, general anesthesia was induced in order to allow studies involving extensive surgery, nerve stimulations, and the like, to be carried out. The general anesthetic used following the local anesthetic was urethane, urethane and ether, ether, or ether and chloroform. The action of these on the pig will be discussed in a later paper. In the present paper the kind of anesthetic used will be mentioned when it is believed that this knowledge will be helpful to the reader.

METHODS AND APPARATUS

The blood-pressure records were taken from the central (heart) end of a ligated carotid artery with either a mercury manometer or a Harvard membrane manometer writing on a long-paper kymograph. Sodium citrate solution (generally 6 per cent) was used to prevent blood clots in the arterial cannula and the tubes.

Hydrostatic effects in the manometric system were obviated where quantitative results were desired.

The respiratory tracings were recorded with a Becker air tambour. The receiving end of the system consisted of either a pneumograph tied around the chest or a hypodermic needle inserted into the trachea.

A Stoelting inductorium with two dry cells in the primary circuit furnished the current for electrical stimulation. The voltage of this circuit was determined on many occasions and found to be about 2.8.

In all experiments time tracings were made. Throughout the course of most experiments involving the use of a general anesthetic, moderate heat was supplied to the animal from the heating mechanism of the Brodie operating-table. When ether or chloroform was used as an anesthetic, the general rule was to pass it through the ether-warmer before admitting it to the lungs of the animal.

Except when gravity effects were being studied, the animals were, during all procedures, kept in a horizontal position, usually on the back. In the open-chest and open-abdomen experiments the animals were laid on the side.

Further details of procedures will be given in appropriate places.

II. The Normal Blood Pressure

As is well known, blood-pressure tracings made with the mercury manometer show at least two distinct kinds of waves: short ones due to the beat of the heart, and long ones due to the respirations. All blood-pressure figures recorded in the text of this paper were determined by measuring vertically from the base line, not to the highest or lowest point on the tracing but to about the mid-point. The figures represent, therefore, approximately the mean blood pressure under the conditions prevailing.

Mean Blood Pressure in the Carotid Artery

In table I we present the results of blood-pressure determinations, with a mercury manometer, on 14 pigs under local anesthesia. All figures represent mean pressures occurring in the quiet animal within a few minutes after inserting the cannula into the carotid artery. Since, as stated above, the artery was ligated, the values must represent both the side pressure and the velocity pressure, and because the distance between the cannula and heart was short, they must approximate the mean pressure in the aorta itself.

TABLE I—*Mean carotid blood pressure under local anesthesia**

PIG	BREED	SEX	WEIGHT (KG.)	MEAN BLOOD PRESSURE (MM. HG)
10	Tamworth	♂ castrate	34	162
14	Tamworth	♂ castrate	30	158
15	Tamworth	♂ castrate	37	144
16	Tamworth	♂ castrate	32	184
18	Tamworth	♂ castrate	30	170
19	Tamworth	♂ castrate	41	185
20	Duroc Jersey	♂ castrate	39	180
21	Tamworth	♂ castrate	43	180
22	Duroc Jersey	♀	33	168
23	Poland China	♀	31	180
24	Poland China	♀	30	176
25	Poland China	♂	19	150
26	Tamworth	♂	33	166
27	Tamworth	♂ castrate	30	160
Averages			33	169

*The local anesthetic was, with one exception, 2 per cent procaine. In the case of pig 10, it was 2 per cent apothesine.

A typical blood-pressure tracing taken under the above-mentioned conditions is shown in figure 1. A blood-pressure tracing taken under similar conditions, but with a membrane manometer, is shown in figure 2.

Discussion: The blood-pressure values presented in table I are believed to be highly significant because of the careful conditions under which they were taken and because none of them deviate markedly from the mean of 169 mm. Hg. The two greatest deviations are seen in pigs 15 and 25, with pressures of 144 and 150, respectively. If we leave these out of consideration for the reason that they appear to be abnormally low, the mean pressure of the series is raised to only 172. There was nothing in the condition of the health of these two pigs to account for the low blood pressures, and we regard the pressures as normal.

That the local anesthetic itself was not responsible for the generally high blood pressures is indicated by experiments, to be described in a later paper, in which it was shown that procaine and apothesine are without effect on blood pressure. Whenever a pig struggled or coughed, the blood pressure rose, but such rises were disregarded. Therefore nothing of that kind can account for the high pressures.

The work of Horning and McKee[8] shows that in dogs there is a positive correlation between blood pressure and body weight. In our pigs there is little evidence of such a relationship. Possibly

it would show up in a group of animals covering a greater range of weight. It is well known that in human beings there is a positive correlation between blood pressure and age. The exact ages of our pigs are not known, but the age-range was a matter of only a few months. Therefore, we would need a greater range before we could decide the influence of age.

In general, the blood-pressure values reported here are somewhat greater than we expected to find, judging by the figures obtained in other animals. In a mature cow, Dukes and Emmerson,[5] using a local anesthetic, found a carotid blood pressure of only 125 mm. Hg. In the carotid artery of several sheep, Dresbach[4] found, under local anesthesia, an average blood pressure of only 110 mm. Hg.

Pulse Pressure

The blood-pressure results reported in table I are mean values. As is well known, the mercury manometer does not give a complete picture of the rapid pressure changes in the circulation, such as those due to the heart beat; it tends rather to give a picture of the mean pressure because of the inertia of the mercury. In an effort to determine the amount of the pulse pressure, that is, the full pressure change due to the heart beat, a membrane manometer, which has a high frequency, was connected to a cannula in the carotid artery of a pig under local anesthesia. A blood-pressure tracing was then made and the membrane manometer was calibrated. Under these conditions the maximum pressure attained during systole of the heart was 200 mm. Hg; the minimum pressure during diastole, 140 mm. Hg. Thus by difference we obtain a pulse pressure of 60 mm. Hg. Even the membrane manometer is not a perfect recorder and the span between the minimum pressure of 140 and the maximum pressure of 200 is probably widened by the fling of the lever. However, we used a light lever, which would tend to minimize the amount of fling.

III. The Influence of Non-Nervous Factors on Blood Pressure

Gravity

To study the influence of gravity on blood pressure, we used a urethanized pig, whose vagi were cut. The animal was secured in the horizontal back-down position in such a way that either end of the animal could be raised or lowered, care being taken to keep the axis of rotation directly under the cannula in the carotid artery.

Rotating the animal so that the head was up and the hind feet down caused the blood pressure to drop from 64 to 26 mm. Hg. Rotating the animal so that the head was down and the hind feet up caused the blood pressure to rise—in one trial, from 70 to 90 mm. Hg; in another, from 70 to 96 mm. Hg (fig. 3).

These results are similar to those reported by other workers for certain other mammals.[1,7]

Compression of the Abdomen

In a pig whose spinal cord had been sectioned at the atlanto-occipital articulation, the effect on the carotid blood pressure of applying a tight abdominal binder was determined. Such compression caused the blood pressure to rise from 50 to 100 mm. Hg. There probably is nothing unusual about this result.

Ligation of Both Carotid Arteries

We studied in the following way the influence of obstruction of both carotid arteries on blood pressure. A cannula was inserted into the central end of a carotid artery in the usual way, that is, after the vessel had been ligated. The other carotid was then ligated or occluded and the immediate effect on the blood pressure noted. In two pigs, one under urethane anesthesia and the other under ether anesthesia, the effect was negative; in a third pig (urethane anesthesia) the fall was only slight—94 to 86 mm. Hg. In a fourth pig (local anesthesia) both carotid arteries were ligated, and the consciousness of the animal seemed reduced but was not abolished. In this pig the blood pressure was not measured before ligation of either artery, but after ligation of both it was normal, that is, 185 mm. Hg.

Comment: It is evident that ligation of both carotid arteries in the pig has little or no immediate effect on blood pressure. In man it is stated that ligation of both carotids is dangerous or fatal.[9]

Asphyxia

In order to determine the effect of asphyxia on the blood pressure of the pig, the trachea of a urethanized animal was completely occluded for an interval of 66 seconds. During the occlusion the blood pressure rose from 70 to 160 mm. Hg (fig. 4). The trachea was then opened and the blood pressure continued to rise, for a short time, to 190 mm. Hg.

Comment: It is well known that asphyxia causes a rise of blood pressure.[1] Evidently this pig was no exception to what has been observed in other animals.

The pig is sometimes regarded as an animal whose cardiovascular mechanism possesses comparatively little reserve power. The great rise of blood pressure seen in this pig under asphyxia does not support such a view.

Relation of Respiratory Waves of Blood Pressure to Respiration

It has long been known that blood pressure undergoes variations with respiration. However, there is no general agreement as to when the changes come in relation to the phases of respiration. That differences occur in different species and even in different individuals is made probable by the work of a number of investigators. In man and the rabbit, most workers find a simple fall with inspiration and a simple rise with expiration, though exceptions are reported. In the dog, much less uniformity is seen; it appears that many combinations may exist.[17]

Our results in the pig are summarized in table II. All of the blood-pressure tracings were made under local anesthesia with a mercury manometer. With pigs 21, 24 and 25, the length of the rubber tubing connecting the artery to the manometer was 33 inches, whereas with the other pigs the length was 46 inches. However, this difference seemed to be unimportant. The respiratory tracings were made with an air tambour. The receiving end of this recording system was either a needle in the trachea or a pneumograph around the thorax.

Table II—*Respiratory variations of blood pressure**

Pig	Inspiration	Expiration	Method of Recording Respirations
14	+	−	Tambour and pneumograph
16	+	−	Tambour and pneumograph
18	+−	−+	Tambour and needle in trachea
19	+−	−+	Tambour and needle in trachea
20	−+	+−	Tambour and needle in trachea
21		+−	Tambour and needle in trachea
24	−+	+−	Tambour and pneumograph
25	+	−	Tambour and pneumograph

*Local anesthesia.
+ = a rise of blood pressure.
− = a fall of blood pressure.

Comment: From these results no rule can be laid down as to the relation of the phase of respiration to the respiratory variation of blood pressure in the pig. The results suggest that the method of recording the respirations is of significance. It would be better

to record the blood pressure with a membrane manometer or some other recorder with a short period.

Figure 5 shows superimposed blood pressure and respiratory tracings.

Curves of Pressure Changes

The character of the pressure changes in the carotid artery was studied in four pigs. The method was to connect a membrane manometer through rubber tubing to a carotid cannula and to take a record of the pressure changes on a rapidly moving smoked paper. The rubber tubing was taped throughout its length to make it inelastic; the inside diameter of the tube was 3 mm.; the length, 35 cm. The diameter of the tip of the arterial cannula was approximately 1.5 mm.

Comment: None of the tracings showed any feature that could be considered characteristic of the pig. Three of the tracings were very much alike. One of these is shown in figure 6. The fourth one showed a catacrotic (descending) limb that, from the apex of the curve to the dicrotic notch, was more nearly vertical than usual.

Adrenalin

The action on blood pressure of solution of adrenalin chlorid (P. D. & Co.), in doses varying from 0.2 to 1 cc, was studied in several animals, under either urethane or ether anesthesia. Some of the results are as follows:

In one pig, under ether anesthesia and with both vagi cut, 1 cc of adrenalin given intravenously caused the blood pressure to rise from 64 to 175 mm. Hg. It returned to normal in about 2.8 minutes. In another pig, under urethane anesthesia and with both vagi cut, 1 cc of adrenalin given intravenously caused the blood pressure to rise from 45 to 180 mm. Hg. The effect was practically gone at the end of 2.5 minutes. In a pig, under urethane anesthesia and with an open thorax and both vagi cut, injection of 1 cc of adrenalin directly into the heart caused the blood pressure to rise from 38 to 94 mm. Hg. In a "spinal" pig the injection of 0.5 cc of adrenalin intravenously caused the blood pressure to rise from 53 to 170 mm. Hg (fig. 7). In the same pig, the injection of 0.5 cc of adrenalin subcutaneously in the belly region gave negative results on blood pressure; and the same dose injected into the muscles of the thigh gave negative results.

Comment: As far as they go, these experiments indicate that the action of adrenalin on the blood pressure of the pig is similar to its action on the blood pressure of other mammals.

As stated above, the pig is sometimes regarded as an animal whose cardiovascular mechanism possesses comparatively little reserve power. It is doubtful if a cardiovascular mechanism that gives such a decided response to adrenalin can be regarded as incompetent.

Pituitrin

The action of pituitary extract on blood pressure was studied in two pigs. The pituitary preparation used was liquor pituitarii (P.-M. Co.), in intravenous doses of 1 cc. In one pig (ether anesthesia) the blood pressure rose from 120 to 146 mm. Hg, returning to normal in about one minute; in another pig (ether anesthesia) the rise was from 106 to 130 mm. Hg, and the return to normal was at the end of two minutes (fig. 8).

There is nothing in the results of these experiments to indicate that the reaction of the blood pressure of the pig to pituitrin is different from the reaction in other mammals.

IV. The Influence of Nervous Factors on Blood Pressure and Heart Rate

The Amount of Vagus Tone as Elicited by Atropin

It is well known that the vagi are the inhibitory nerves of the heart and that their action (except in small animals, *e. g.*, mouse, rat, guinea pig[3]) is tonic or continuous. The increase of heart rate following abolishing vagus control is directly related to the reserve power of the heart.

Some of our experiments on the effect of tightly tying or cutting the vagi, which of course removes the tonic influence, did not give the anticipated increases in heart rate. Therefore, in order to study the amount of vagus tone under more favorable conditions, we resorted to the use of atropin injections in unanesthetized pigs. The final effect of atropin on the heart is to paralyze the endings of the vagus nerves. The heart rate then increases because the tonic influence of the vagi is removed.

The effect of atropin injection on the heart rate is shown in table III.

Table III—*Effect of atropin injection (1/50 grain) on heart rate of pig*

Pig	Weight (kg.)	Average Heart Rate Before Atropin	Maximum Heart Rate After Atropin	Increase (%)
11	49	146	203	39
12	57	155	188	21
13	40	129	212	64

Comment: Using the increase in heart rate following injection of atropin as the index of vagus tone, it appears from table III that such tone is fairly well developed.

Amount of Vagus Tone as Elicited by Tightly Tying or Cutting the Vagus

The effect on the heart rate of tightly ligating or cutting a vagus, as studied in several animals, was not constant. Sometimes the heart rate was increased and sometimes it was not. The most definite increases of heart rate obtained when one vagus was tightly tied (other vagus intact) are as follows: 85 per cent, 60 per cent, 20 per cent. There seemed to be no definite relation between the kind of anesthetic used and the nature of the response of the heart when a vagus was tied.

Comment: The atropin experiments outlined above reveal vagus tone in all cases. The experiments on tying or cutting the vagus reveal tone in some cases and not in others. Further studies of the effect of tying or cutting the vagi under more rigidly controlled conditions are necessary before we are willing to say that even a small percentage of pigs lack tonic vagus inhibition of the heart. Age has been shown to be an important factor in certain other mammals, and it might be of importance in the pig.

Stimulation of Peripheral End of Vagus

The peripheral (heart) end of each vagus was stimulated on many occasions in many animals. In table IV we have compiled the results on blood pressure and heart rate of all stimulations (24 in number) when the distance between the primary and secondary coil was 10 cm. This makes the results comparable because the strength of stimulus was the same.

Table IV—*Effect of stimulation of peripheral end of vagus on blood pressure and heart (coil-distance 10 cm.)*

	Number of Stimulations Causing				Number of Animals Studied
	Fall of B.P. and Complete Inhibition of Heart	Fall of B.P. and Slowing of Heart	Slowing of Heart Only	Negative Effect	
Right vagus	7	8	1	0	15
Left vagus	0	5	2	1	7

B. P. = blood pressure.

Typical results obtained with each nerve are shown in figures 9 and 10.

Discussion: From a survey of the results of stimulation of the peripheral end of the vagus (table IV), it is apparent that inhibition was more highly developed in the right nerve of the animals studied than in the left. Attention is directed to the fact that left-vagus stimulation did not cause complete heart inhibition on any occasion. Fifteen other stimulations of the left vagus, at varying coil-distances, failed in each instance to cause complete inhibition. On four other occasions complete inhibition was obtained by stimulating the peripheral end of the right nerve. There can be no question that inhibition is better developed in the right nerve. This is in conformity with the general condition in other animals.[17]

Does the Cervical Vagus Nerve Contain Cardiac Accelerator Fibers?

It has been taught from time to time[9] that cardiac accelerator fibers are contained in the cervical part of the vagus nerve and that when the inhibitory terminations in the heart are paralyzed by atropin, stimulation of the peripheral end of the vagus causes cardiac acceleration. Hering[6] denies the existence of accelerators in the vagus of the dog, cat, rabbit and monkey, and we obtained negative results in a pig. Following an injection of atropin, repeated stimulation of the peripheral end of the right vagus nerve failed to cause acceleration of the heart.

Stimulation of the Central End of the Vagus

The effects of stimulation of the central (head) end of the vagus nerves on blood pressure are shown in table V.

Table V—*Effect of stimulation of central end of vagus nerve on blood pressure (coil-distance 10 cm.)*

	Number of Stimulations Causing						Number of Animals Studied
	Rise		Fall		Negative Effect		
	Other Vagus Intact	Other Vagus Cut	Other Vagus Intact	Other Vagus Cut	Other Vagus Intact	Other Vagus Cut	
Right vagus	10	5	2	0	0	1	11
Left vagus	0	5	1	1	1	3	10

Discussion: When the central end of a nerve is stimulated the blood pressure may rise or fall, depending on whether pressor or depressor nerve fibers are affected. The tendency to rise is

greater than the tendency to fall. Negative effects are sometimes seen.

The results in table V show that in the pig the tendency of the blood pressure to rise when either vagus is stimulated is greater than its tendency to fall, and that negative results are obtained more often with the left vagus than with the right vagus.

Out of 43 additional stimulations (of various strengths) of the central end of the vagus, both right and left, there were only three instances in which the blood pressure fell, these being confined to the left nerve (right nerve intact). In the great majority of these 43 stimulations the blood pressure rose; in a few instances no effect was obtained.

Evidently, then, a pressor response to stimulation of the central end of the vagus nerves was the rule in these experiments. Depressor effects, when obtained, usually occurred when the other vagus was intact. Figures 11 and 12 illustrate pressor responses. Figure 13 illustrates a depressor response.

Thirteen pigs were used in this study. Urethane anesthesia was used in the majority of animals; ether anesthesia in the others. The kind of response seemed to bear no relation to the kind of anesthetic.

As will be discussed below, we would find no experimental evidence of the existence of a separate depressor nerve in the pig. One would expect, therefore, that the depressor fibers would be incorporated in the vagus trunk and that depressor effects might be obtained fairly frequently when the central end of this trunk is stimulated. Our results show, however, that depressor responses are infrequent.

The Question of a Separate Depressor Nerve

In the rabbit the depressor nerve is a separate entity. In the pig it is said to be separate.[12] In most animals the depressor fibers are carried in the trunk of the vagus. In the rabbit, stimulation of the depressor nerve causes vascular dilatation and cardiac inhibition. In animals that have the depressor nerve incorporated in the vagus trunk, stimulation of the central end of the vagus may at times cause a fall in blood pressure.

In several pigs we studied the question of the existence of a separate depressor nerve. While we found, on one or two occasions, nerve strands that were very suggestive anatomically, we were unable by electrical stimulation to obtain any evidence of the existence of a separate depressor nerve.

Stimulation of the Cervical Sympathetic Nerve

In the pig the cervical sympathetic nerve is separate from the vagus on both sides. In our earlier experiments on vagus stimulation no attention was given to the cervical sympathetic and probably at times it was stimulated along with the vagus and at times it was not. In our later experiments the vagus and sympathetic were carefully separated on both sides before the vagus was stimulated; and in order to control previous experiments in which the separation was not made, we stimulated both the peripheral and the central ends of the cervical sympathetic on each side in several animals, and observed the effect on blood pressure. Stimulation of the central end gave a negative effect, except in one instance where a slight rise was obtained. Stimulation of the peripheral end usually gave a slight rise, though a slight fall and a slight rise and fall also were obtained. In no case was the blood pressure response to stimulation of either end of the cervical sympathetic nerve great enough to alter the conclusions drawn from any previous experiment involving stimulation of a vagus nerve unseparated from the cervical sympathetic.

Stimulation of the Central End of the Anterior Laryngeal Nerve

In one pig under urethane anesthesia the central end of an anterior laryngeal nerve was stimulated at coil-distances of 15, 10, 5 and 0. In all cases the effect on blood pressure was, contrary to our expectations, so slight as to be of little or no significance.

Stimulation of the Central End of the Sciatic Nerve

The effects of stimulation of the central end of the sciatic nerve on blood pressure are shown in table VI. Both sciatic nerves were at one time or another stimulated. No difference in the response of the two nerves was noted. The coil-distance was 10 cm. Four of the animals were anesthetized with urethane, three with ether, and one with urethane and ether.

Table VI—*Effect of stimulation of central end of sciatic nerve on blood pressure (coil-distance 10 cm.)*

Number of Stimulations Causing			Number of Animals Studied
Rise	Fall	Little or No Effect	
6	1	2	8

Table VII is similar to table VI except that the coil-distance was a variable (15, 5, 0 cm.).

TABLE VII—*Effect of stimulation of central end of sciatic nerve on blood pressure (coil-distance variable)*

Number of Stimulations Causing				Number of Animals Studied
Rise	Fall	Rise and Fall	Little or No Effect	
10	1	3	6	8

Comment: The results summarized in tables VI and VII indicate that stimulation of the central end of the sciatic nerve usually causes a rise in blood pressure, but that negative effects are not uncommon. A rise and a fall, as a result of the same stimulus, is not common; a pure fall is rare. Figure 14 illustrates a rise in blood pressure caused by stimulation of the central end of the sciatic nerve.

No relation was seen to exist between the nature of the response and the kind of anesthetic.

Cardiac Acceleration and Augmentation

The accelerator nerves pass to the heart in the cardiac plexus. In order to stimulate a branch of the plexus, it is necessary to open the thorax of the animal. After this, artificial respiration must of course be used. It was necessary to make attempts on several pigs before we were able to elicit heart acceleration by stimulating a branch of the cardiac plexus. Figure 15 portrays the results of a successful experiment. The branch of the plexus (left) was divided so as to prevent possible reflex effects, and the peripheral end was stimulated. The coil-distance was 10 cm.

In another pig an augmentor effect without acceleration was obtained by stimulating a branch of the cardiac plexus (fig. 16). Because the branch was not divided prior to stimulation, the possibility remains that the effect was a reflex one. However, this is improbable.

In still another pig good acceleration of the heart was repeatedly provoked by stimulating a point on the heart a half-inch below the entrance of the anterior vena cava (fig. 17). This point is undoubtedly in the region of the sino-auricular node.

It may be of interest to record that in a pig stimulation of the posterior cervical and first thoracic ganglia (right) and all their branches (uncut) failed to cause cardiac acceleration. In the

same pig stimulation of the posterior cervical ganglion caused cardiac inhibition (fig. 18).

Stimulation of the Splanchnic Nerve

The splanchnic nerve carries vasoconstrictor fibers to the abdominal viscera. We investigated the effect of stimulation of the peripheral end of the divided left splanchnic nerve in several pigs, and in every case a distinct rise of blood pressure was obtained. In one animal with a very low blood pressure, rises of nearly 100 per cent were observed. A typical result is shown in figure 19.

The central end of the splanchnic nerve was stimulated twice in a pig and each time a slight rise of blood pressure was obtained. These effects of splanchnic stimulation are not considered to be unusual.

Stimulation of the Sympathetic Trunk

In a urethanized pig the central end of the right sympathetic trunk was stimulated opposite the fourteenth rib, and the blood pressure rose from 90 to 152 mm. Hg (fig. 20).

Tone of the Vasoconstrictor Center

In order to obtain some idea about the amount of tonic control exerted by the vasoconstrictor center on the peripheral blood-vessels, we sectioned the spinal cord just below the medulla in several pigs. Following the section, artificial respiration was given. In one pig, under ether anesthesia, sectioning the cord caused the blood pressure to drop to less than one-half its former level, that is, from 86 to 40 mm. Hg (fig. 21).

In a pig under urethane anesthesia, the fall was from 68 to 24 mm. Hg; and in another pig under urethane the fall was from 36 to 32 mm. Hg.

In the first-mentioned pig, following division of the cord, we maintained the animal under artificial respiration for a three-hour period, during which time the blood pressure rose only 10 mm. Hg. Care was taken to prevent a fall in body temperature by using artificial heat. The object of this experiment was to see to what extent the blood pressure is able to rise after the blood-vessels are freed from control of the medulla.

Comment: These experiments indicate that the vasoconstrictor center of the pig possesses appreciable tone, as judged by the fall in blood pressure following section of the cord. The last part of the experiment indicates that the spinal cord and blood-vessels are unable by themselves to restore a fallen blood pressure.

V. Summary

The lack of information in the pig on the blood pressure and its relations, and on the cardiovascular mechanism generally, is noted. A plea is made for the development of a special physiology of farm animals.

The mean blood pressure in the ligated carotid artery of 14 pigs under local anesthesia was determined, the average for the group being 169 mm. Hg.

The effect of gravity on the blood pressure of the pig was found to be similar to the effect reported by other workers for certain other animals.

Compression of the abdomen caused a rise in blood pressure similar to what might be expected.

Ligation of both carotid arteries caused little or no effect (immediate) on blood pressure. In a pig under local anesthesia, ligation of both carotid arteries appeared to reduce but did not abolish consciousness.

Asphyxia caused a sharp rise in blood pressure.

From our results no general rule can be laid down as to the relation of respiration to respiratory waves of blood pressure.

The curves of pressure changes in the carotid artery present no feature that could be said to be characteristic of the pig.

As far as the evidence goes, the action of adrenalin and pituitrin on the blood pressure of the pig is similar to their action in other species.

The balance of evidence indicates that vagus tone is fairly well developed in the pig.

Cardiac inhibitory action was found to be better developed in the right vagus nerve than in the left.

In a pig we found no evidence of the existence of accelerator fibers in the cervical vagus nerve (right).

Stimulation of the central end of the vagus nerve caused a rise of blood pressure more often than a fall. Negative effects were more common with the left nerve.

No experimental evidence of the existence of a separate depressor nerve was obtained in studies involving several pigs.

In the pig the cervical sympathetic nerve on both sides is separate from the vagus. Stimulation of either end of the cervical sympathetic nerve gave negative or only slight effects on blood pressure.

Stimulation of the central end of the sciatic nerve usually gave

a rise in blood pressure, although negative results were not uncommon. A fall was seldom obtained.

Acceleration and augmentation of the heart beat were obtained from stimulation of the cardiac plexus of the pig. Stimulation in the region of the sino-auricular node caused cardiac acceleration.

Stimulation of the peripheral end of the splanchnic nerve caused a rise of blood pressure, in some instances as great as 100 per cent. Stimulation of the central end of the splanchnic nerve caused a rise of blood pressure.

Stimulation of the central end of the sympathetic trunk (right) opposite the fourteenth rib caused a sharp rise of blood pressure.

The vasoconstrictor center of the pig possesses appreciable tone.

Bibliography

[1]Bayliss, W. M.: The Vasomotor System (Longmans, Green and Co., London, 1923).
[2]Bethe, A., Bergmann, G. von, Embden, G., Ellenger, A.: Handbuch der normalen und pathologischen Physiologie, (Julius Springer, Berlin, 1927), VII.
[3]Clark, A. J.: The Comparative Physiology of the Heart (Cambridge University Press, 1927).
[4]Dresbach, M.: Observations upon the blood pressure of sheep. Amer. Jour. Physiol., xxv (1910), p. 433.
[5]Dukes, H. H., and Emmerson, M. A.: Unpublished results.
[6]Hering, H. E.: Uber das angebliche Vorkommen von Acceleransfasers im Halsvagus der Säugetiere. Arch. f. d. ges. Physiol., cciii (1924), p. 100.
[7]Hill, L.: The influence of the force of gravity on the circulation of the blood. Jour. Physiol., xviii (1895), p. 15.
[8]Horning, J. G., and McKee, A. J.: Blood pressure and its application in canine practice. Jour. A. V. M. A., lxviii (1925), p. 221.
[9]Howell, W. M.: A Textbook of Physiology (10th ed., W. B. Saunders Co., Philadelphia and London, 1927).
[10]Kuntz, A.: The Autonomic Nervous System (Lea and Febiger, Philadelphia, 1929).
[11]Marey, E. J.: La Circulation du Sang (Paris, 1881).
[12]Martin, P.: Lehrbuch der Anatomie der Haustiere, (Stuttgart, 1922), IV.
[13]Ranson, S. W.: Afferent paths for visceral reflexes. Physiol. Rev., i (1921), p. 477.
[14]Sherrington, C. S.: Mammalian Physiology (Humphrey Milford, Oxford, 1919).
[15]Tigerstedt, R.: Die Physiologie des Kreislaufes, (2nd ed., Walter de Gruyter & Co., Berlin and Leipzig, 1921-1923), I-IV.
[16]Wiggers, C. J.: The Pressure Pulses in the Cardiovascular System (Longmans, Green and Co., London and New York, 1928).
[17]Wiggers, C. J.: The Circulation in Health and Disease (2nd ed., Lea and Febiger, Philadelphia and London, 1923).

Descriptions of Figures

Figure 1. Blood-pressure tracing from the carotid artery of a 33-kg. pig. Local anesthesia. Vagi intact. Hg manometer. Note the marked respiratory variations in pressure. These were commonly found in animals under local anesthesia and are believed to be normal.

Figure 2. Blood-pressure tracing from carotid artery, membrane manometer. 43-kg. pig. Local anesthesia. B. P., carotid blood pressure. Calibration is in terms of mm. Hg.

Figure 3. Effect of gravity on blood pressure. 22-kg. pig. Urethane anesthesia. Vagi cut. B. P., carotid blood pressure, Hg manometer; B. L., line of zero arterial pressure and signal line; H. D., head down; H. U., head up.

Figure 4. Effect of asphyxia on blood pressure. 22-kg. pig. Urethane anesthesia. Vagi cut. B. P., carotid blood pressure, Hg manometer; B. L., line of zero arterial pressure and signal line. Trachea occluded between signals. Note cessation of breathing at a.

DESCRIPTIONS OF FIGURES—*Continued*

Figure 5. Relation of respirations to respiratory waves of blood pressure. 43-kg. pig. Local anesthesia. Resp., respirations (needle in trachea and tambour, hence down-stroke = inspiration); B. P., carotid blood pressure, Hg manometer; B. L., line of zero arterial pressure. The writing points were practically perfectly superimposed. Note that the blood pressure rises with expiration and falls during the pause after expiration. For other relations and comments, see text.

Figure 6. Blood-pressure tracing from the carotid artery, membrane manometer. 43-kg. pig. Local anesthesia. B. P., carotid blood pressure, fast drums. Shows character of pressure pulse.

Figure 7. Effect of adrenalin chlorid on blood pressure. 30-kg. pig. "Spinal" preparation. B. P., carotid blood pressure, Hg manometer; B. L., line of zero arterial pressure and signal line. At second signal, 0.5 cc adrenalin chlorid solution (P. D. & Co.) was given intravenously. Note very sharp rise of blood pressure and absence of vagus inhibition.

Figure 8. Effect of pituitary extract on blood pressure. 31-kg. pig. Ether anesthesia. Vagi intact. B. P., carotid blood pressure, Hg manometer; B. L., line of zero arterial pressure and signal line. At signal, 1 cc of solution of pituitary (P.-M. Co.) was given intravenously.

Figure 9. Effect of stimulation of peripheral end of right vagus nerve on blood pressure and heart beat. 32-kg. pig. Ether and chloroform anesthesia. B. P., carotid blood pressure, membrane manometer; P. R. Vag. 10, stimulation of peripheral end of right vagus, 10 cm.

Figure 10. Effect of stimulation of peripheral end of left vagus nerve on blood pressure. 40-kg. pig. Urethane anesthesia. B. P., carotid blood pressure, Hg manometer; B. L., line of zero arterial pressure and signal line. At the signal, the peripheral end of left vagus was stimulated, 10 cm. Note absence of complete inhibition of heart.

Figure 11. Effect of stimulation of central end of right vagus nerve on blood pressure. 33-kg. pig. Urethane anesthesia. Left vagus intact. B. P., carotid blood pressure, Hg manometer; B. L., line of zero arterial pressure and signal line. At the signal, central end of right vagus was stimulated, 10 cm.

Fig. 12. Effect of stimulation of central end of left vagus nerve on blood pressure. 37-kg. pig. Ether anesthesia. Right vagus intact. B. P., carotid blood pressure, membrane manometer; C. L. V. 10, stimulation of central end of left vagus, 10 cm.

Figure 13. Effect of stimulation of central end of left vagus nerve on blood pressure and respiration. 35-kg. pig. Ether anesthesia. Resp., respirations; B. P., carotid blood pressure, Hg manometer; B. L., line of zero arterial pressure and signal line. Note fall in blood pressure and complete inhibition of respiration.

Fig. 14. Effect of stimulation of the central end of sciatic nerve on blood pressure. 22-kg. pig. Urethane anesthesia. Vagi cut. B. P., carotid blood pressure, Hg manometer; B. L., line of zero arterial pressure and signal line; C. E. Sci. 10 cm., stimulation of central end of sciatic nerve, 10 cm.

Figure 15. Effect of stimulation of accelerator branch of cardiac plexus on heart rate. 30-kg. pig. Urethane anesthesia. B. P., carotid blood pressure, membrane manometer; L. Accel. 10, stimulation of peripheral end of accelerator branch of cardiac plexus on left side, 10 cm. Note acceleration of heart rate and gradual return to normal.

Figure 16. Effect on heart of stimulation of branch of cardiac plexus. 32-kg. pig. "Spinal" preparation. B. P., carotid blood pressure, membrane manometer; Augment. 10, stimulation of (undivided) branch of cardiac plexus on left side, 10 cm. Note augmentation of heart beat as indicated by increase of pulse pressure.

Figure 17. Effect on heart of stimulation in region of sino-auricular node. 41-kg. pig. Urethane anesthesia. Artificial respiration. B. P., carotid blood pressure, membrane manometer; S-A. N., stimulation of point on heart a half-inch below entrance of anterior vena cava. Note acceleration of heart rate.

DESCRIPTIONS OF FIGURES—*Continued*

Figure 18. Effect on heart of stimulation of posterior cervical ganglion. 33-kg. pig. Urethane anesthesia. Artificial respiration. B. P., carotid blood pressure, membrane manometer; P. C. G. 10, stimulation of posterior cervical ganglion, 10 cm. Note slowing of heart and increase in pulse pressure.

Figure 19. Effect of stimulation of the peripheral end of the splanchnic nerve on blood pressure. 33-kg. pig. Urethane anesthesia. B. P., carotid blood pressure, Hg manometer; B. L., line of zero arterial pressure and signal line. At the signal, peripheral end of left splanchnic nerve was stimulated, 10 cm.

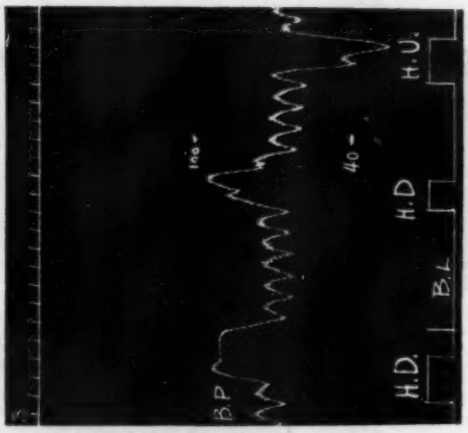


FIG. 3.

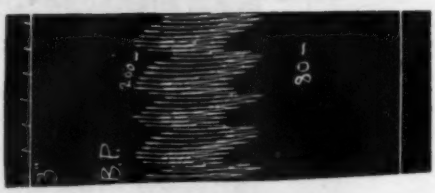


FIG. 2.

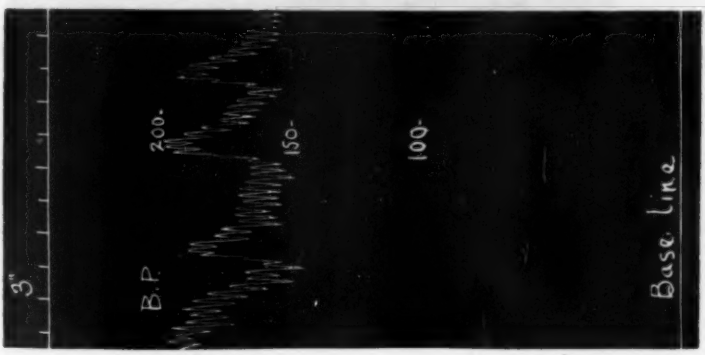


FIG. 1.

DESCRIPTIONS OF FIGURES—*Concluded*

Figure 20. Effect of stimulation of central end of sympathetic trunk on blood pressure. 40-kg. pig. Urethane anesthesia. B. P., carotid blood pressure, Hg manometer; B. L., line of zero arterial pressure. At the signal, central end of right sympathetic trunk was stimulated at level of fourteenth rib.

Figure 21. Effect of dividing the spinal cord on blood pressure. 30-kg. pig. Ether anesthesia. B. P., carotid blood pressure, Hg manometer; B. L., line of zero arterial pressure and signal line. At signals, spinal cord was severed, *i.e.*, in two operations. Artificial respiration begun at c.

FIG. 5.

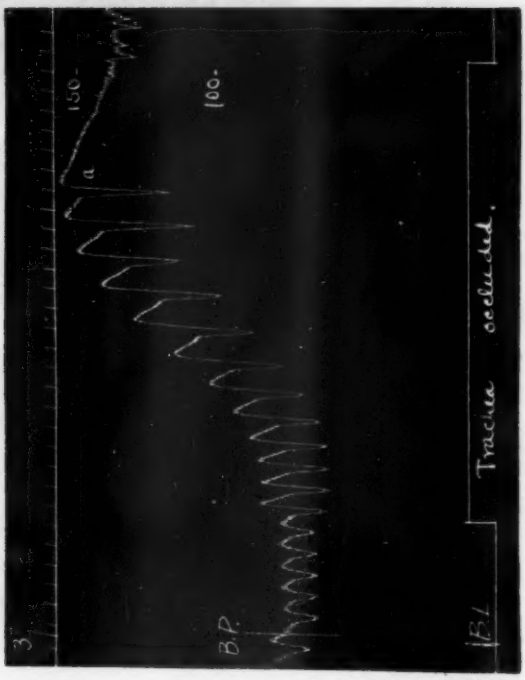
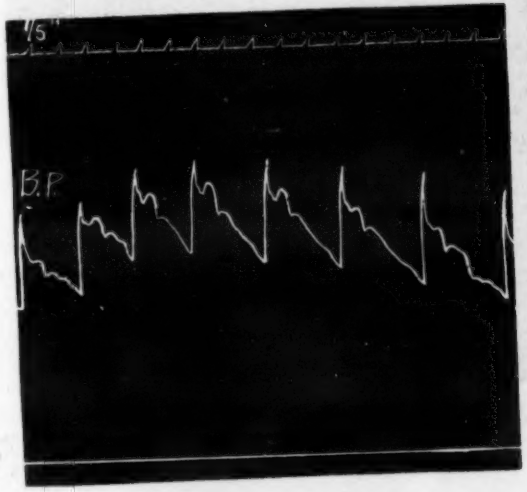


FIG. 4.

Fig. 4.

Fig. 5.



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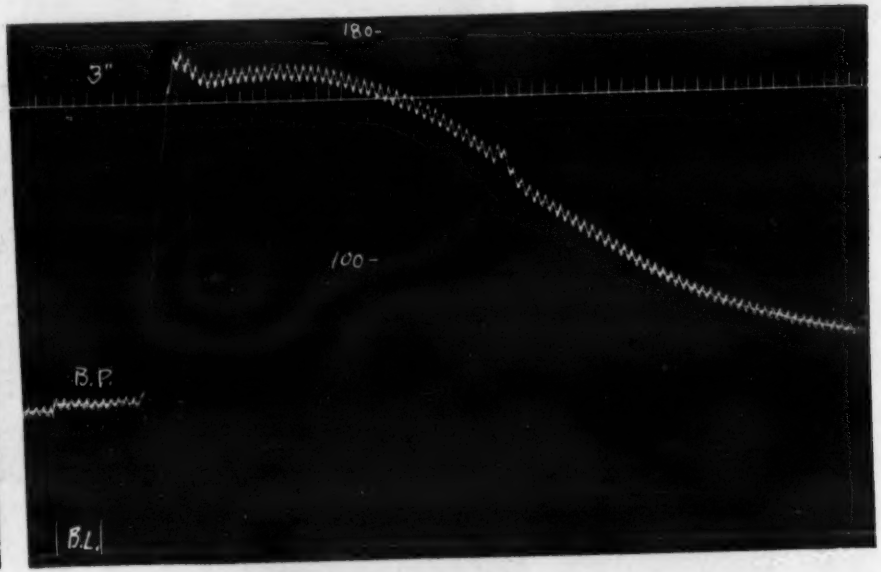

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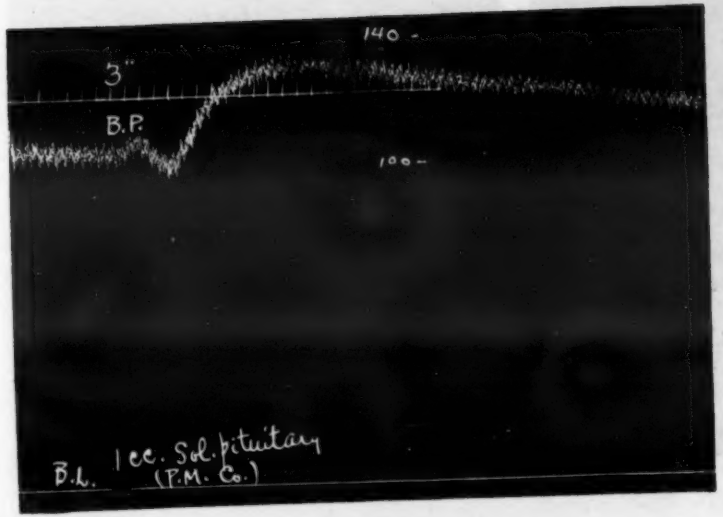

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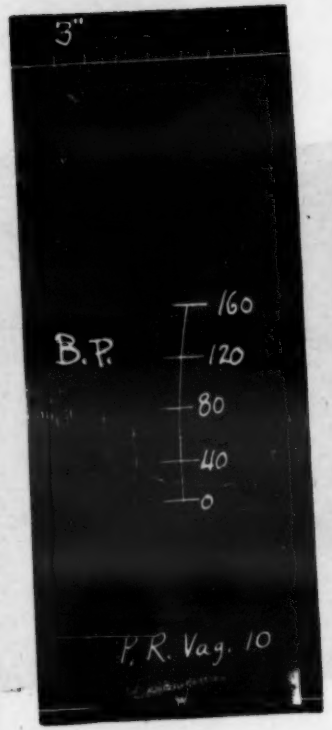

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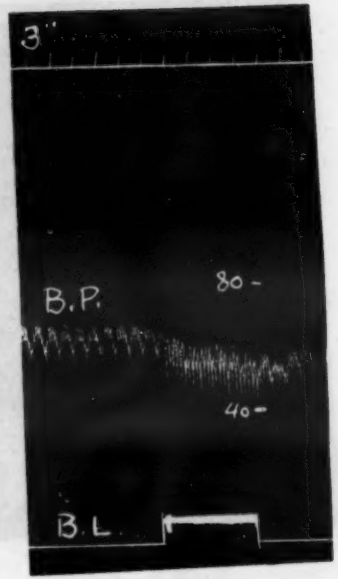

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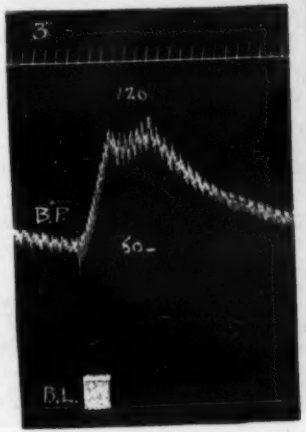
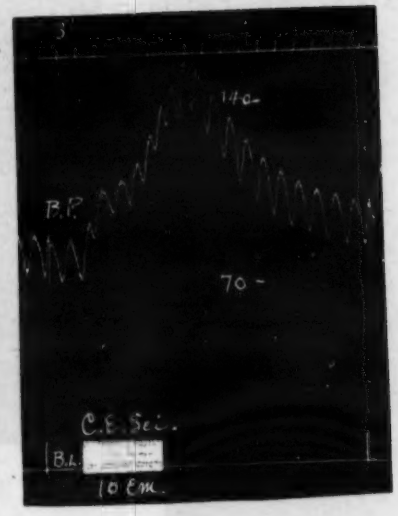


FIG. 11.

FIG. 12.

FIG. 13.



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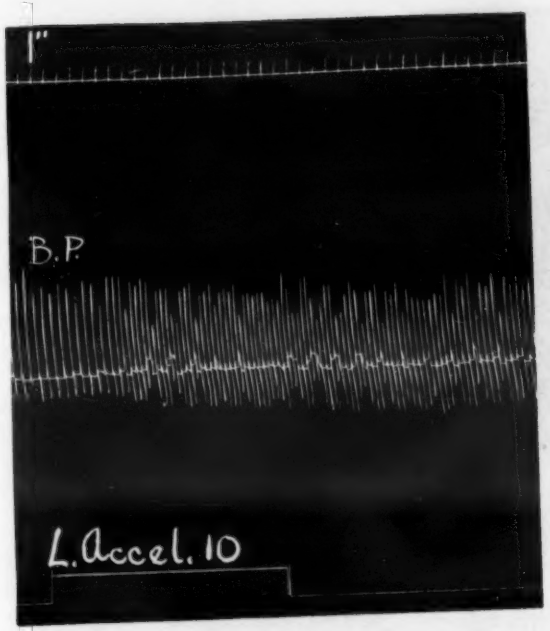
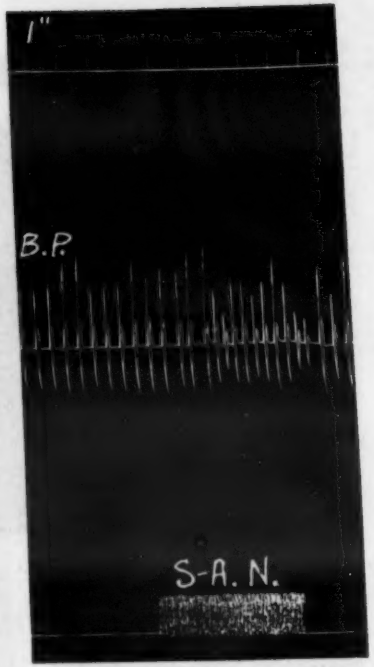


FIG. 15.

FIG. 16.



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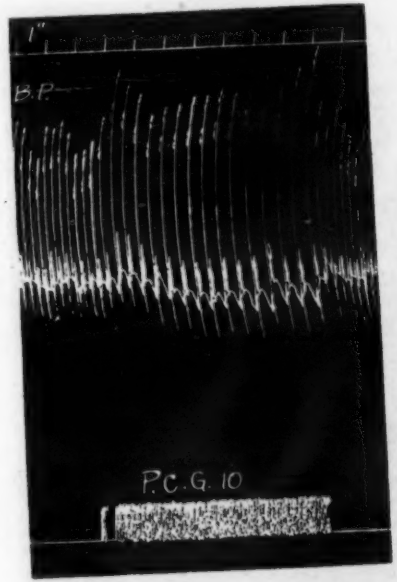

FIG. 18.

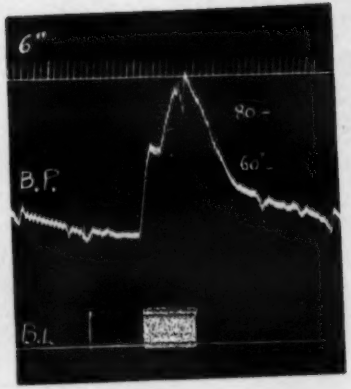

FIG. 19.

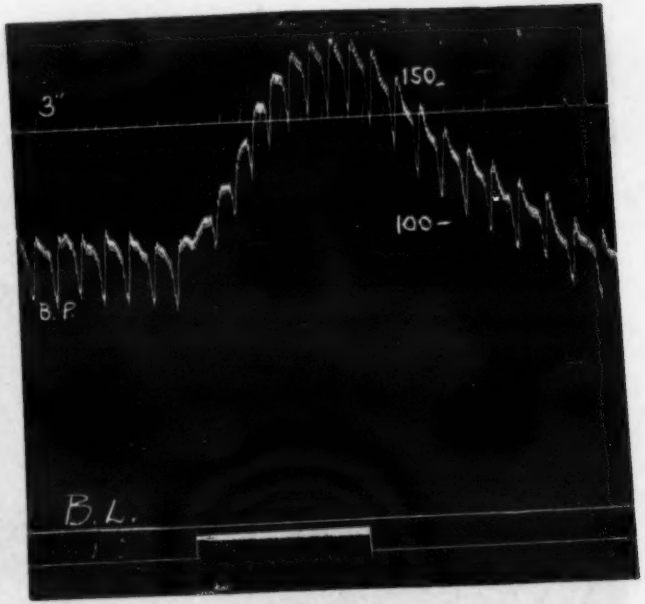

FIG. 20.

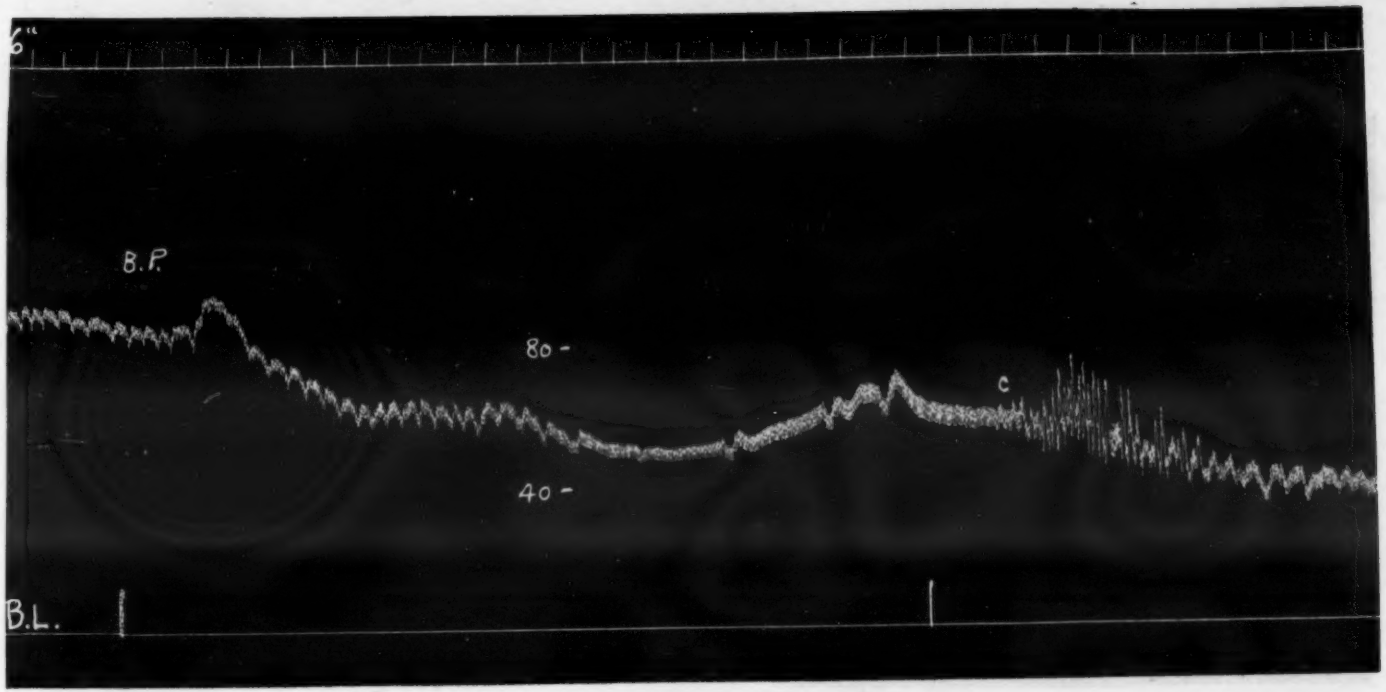

Fig. 21.

A CRITICAL STUDY OF THE BRUCELLA AGGLUTINATION REACTION AND ABORTION RATE IN A HERD OF CATTLE UNDER NATURAL CONDITIONS*

By I. FOREST HUDDLESON, *Bacteriological Section, Michigan State Experiment Station, East Lansing, Mich., and* L. H. SMITH, *Detroit Creamery Farms, Mount Clemens, Mich.*

The natural course of Bang's disease in cattle can be determined only by making a systematic study of a large group of naturally infected and non-infected cattle maintained together under natural conditions for a long period of time. Much may be learned about the trend of the agglutination reaction in those animals that show Brucella agglutinins in their blood serum and about the significance, in terms of active or passive infection, of the reactions in the various agglutination titres one usually employs.

It is also of interest to compare the abortion rate of the negative animals, that is, those that persistently show no evidence of agglutinins in one or more standard dilutions of the blood serum, with those that show agglutinins in one or more dilutions of serum.

Early in 1923, the writers began the compilation of serological and breeding data on a large herd of cattle consisting of pure-bred and grade Holstein, Jersey and Guernsey cattle. We are, therefore, presenting the compiled records of the serological tests and abortions occurring in a total of 541 animals from 1923 up to the present time. Not all of the animals on which data are presented have been in the herd during the entire period. Many have been sold and many have been added during the period which these records cover. The data include only those animals that were tested two or more times at wide intervals.

It is regretted that a systematic study of the causes of all the abortions occurring in this herd was not made. However, a sufficient number of fetuses have been examined bacteriologically at different times during the eight-year period to state definitely that *Brucella abortus*, streptococci, *Bacillus coli* and a vibrio, separately, were the causes of abortions.

During the first four years the animals were tested, the test-tube agglutination method was employed. The rapid method

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was used during the second four years. Serum dilutions of 1:25, 1:50, 1:100, 1:200 and 1:500 were used in the test-tube and serum amounts of 0.08, 0.04, 0.02, 0.01 and 0.004 cc were used in the rapid method. These amounts of serum represent the above respective test-tube dilutions. The results of the agglutination tests on the entire group of animals for the eight test-years, with the exception of those that were always negative, are recorded in table I. The five signs in the year columns after each animal represent the five respective dilutions of serum just referred to. The minus sign (−) represents absence of agglutination; T, trace; P, incomplete and +, complete agglutination.

In analyzing the results of the agglutination test on these animals we find that, of the total, 143 have never shown even a trace of agglutinins in the lowest dilution used. In 151, there have never been sufficient agglutinins to cause more than barely a clumping of the antigen in a 1:25 dilution. We have recorded this type of agglutination as "trace" and "incomplete." It is a question whether all agglutination reactions of this nature are specific or non-specific. Many of them must be of a specific nature because many animals that later show agglutinins in a high titre, at first showed only a slight evidence of agglutinins. As a rule most laboratories, including ours, classify such reactions as negative, and would place them in the negative group. Since these animals have shown evidence of agglutinins, we have separated them from the negative group. There were 26 showing complete agglutination in the 1:25 dilution, 32 in the 1:50 dilution, 24 in the 1:100 dilution, and 165 in the 1:200 and 1:500 dilutions. Those showing reactions in the two latter dilutions are grouped together in the summary, because many of the 1:200 reactors showed almost complete clumping in the 1:500 dilution.

Many of the reacting and negative animals were housed in the same barns until the fall of 1928, when measures were instituted to separate the negative animals from those reacting in a 1:25 dilution or above, by placing them in separate barns. This separation is being continued at the present time. We may assume, therefore, that until the fall of 1928 there was more or less of an opportunity for negative animals to become exposed to infective material from infected animals and for all animals to develop high agglutination titres as a result of the exposure.

At all times good breeding hygiene has been one of the features in the maintenance of this herd. They were under close observation at all times. If an animal showed symptoms of premature

TABLE I—*Trend of Brucella agglutination reactions in cattle over an eight-year period*

Animal	6-23	5-24	6-25	6-27	2-28	11-28	2-29	4-29	7-29	9-29	11-29	2-30	4-30	5-30
6	+ + – – –			+ + + + +		+ + + + +								+ + + + +
21	+ + + + +			P P T – –			+ + + + P				+ + P T –			
24	– – – – –			P T – – –			P T – – –			P T – – –				
25	– – – – –	P – – – –												
27	+ + + + +	+ + + + +		+ + + + +			+ + + + P							
28	– – – – –	– – – – –		– – – – –			P T – – –			P T – – –				
32	+ + + + +	+ + + + P		+ P – – –			T – – – –			T – – – –				– – – – –
33	+ + + + +	+ + + + +		+ + + + +										
35	+ + + + +	+ + + + +		+ + + P T			P P T – –							
36	+ + + + +			+ + P – –										
40	– – – – –	– – – – –		– – – – –						T T – – –		+ + + + +		
44	+ + + + +			+ + + P T			+ + P T –							
53	– – – – –	P P – – –	– – – – –	– – – – –			+ + P T T		+ + P T –	+ + P T –				
55	+ + – – –		+ + + P –	– – – – –			T – – – –							
62	+ + + + +	+ + + + +		+ + + + P			+ + P P T							
63	– – – – –	+ + + P T		– – – – –					T T – – –	– – – – –		– – – – –		
64	+ + + + +			+ + + + +			+ + + P T							
67	– – – – –	– – – – –		T – – – –			T – – – –	T – – – –		– – – – –				
68	– – – – –	– – – – –	– – – – –	– – – – –			T – – – –			– – – – –		– – – – –		
69	+ + + + +			+ + + P T						+ + + + P	+ + + P T			+ + + P T
73	– – – – –	– – – – –	– – – – –	– – – – –				P P T – –						
74	+ + + + +			+ + + P T			+ P T – –			+ P T – –	+ P T – –			+ P T – –
76	– – – – –			+ + + + +			+ + + + +							
77	+ + + – –	+ + + + –		+ + + P T			+ + + + P							
78	+ + + P –			P T – – –			+ + P T –	+ P P T –		+ + P T –	+ + P T –			
79	– – – – –	– – – – –		+ + + + +			+ + + + +							
81	– – – – –	– – – – –		– – – – –		+ P – – –	+ P T – –	+ + P T –		+ + P T –	+ + + P T			
82	– – – – –	– – – – –		– – – – –			T – – – –			– – – – –				
83	– – – – –	– – – – –		– – – – –		– – – – –				– – – – –		T – – – –		
84	– – – – –			– – – – –		– – – – –		T – – – –		– – – – –			– – – – –	
85	+ + + + +	+ + + + +		+ + + + +			+ + + + P							
86	+ + + + +		+ + + + +	– – – – –			T – – – –			T – – – –				
87	+ + + + +		+ + + P –	P T T – –			P T – – –			P T – – –		P T – – –		
88	– – – – –		– – – – –				+ + P T T							
89	+ + + + +			P T T – –			+ + P T –			+ + + P T				
90	+ + + + +	+ P T – –		P – – – –			T – – – –			P T – – –		– – – – –	T – – – –	
91	+ + + + +			– – – – –			P T – – –			P T – – –		– – – – –		
92	+ + + + +		+ + + + +	+ + + + +			+ + + + P			+ + + + +				
93	– – – – –	– – – – –		– – – – –			T – – – –		– – – – –	– – – – –				
94	– – – – –			+ P P – –		+ + P T –				+ + + P T				
95	– – – – –	+ + P – –		P T – – –			P T – – –	P P T – –						
96	– – – – –		– – – – –	– – – – –			– – – – –			– – – – –		– – – – –		T – – – –
100	– – – – –		– – – – –	– – – – –			– – – – –			+ P T – –				
101	+ + – – –			– – – – –			T – – – –			P T – – –		– – – – –		
102	+ + + + +		+ + + + +	+ + + + +			+ + + P T							
103	– – – – –		+ + + + +	+ + + + +			+ + + + +							

TABLE I—*Trend of Brucella agglutination reactions in cattle over an eight-year period—Continued*

Animal	6-23	5-24	6-25	6-27	2-28	11-28	2-29	4-29	7-29	9-29	11-29	2-30	4-30	5-30
118	++ + – –		P – – – –	– – – – –			T – – – –	– – – – –		– – – – –				– – – – –
119	– – – – –		+ + + + +	+ + + P P			+ + + + +	+ + + + +				+ + + + P		
121	– – – – –			+ + + + + +		+ + P P T				+ + + + +		+ + + P T		
124	– – – – –		+ + + P P	– – – – –		T – – – –	T – – – –			T – – –				– – – – –
125	+ + + + +			+ + + + +			T – – – –							
126	– – – – –			+ + + + +			+ P P T –			+ + + P T			P P T – –	
148	+ + + + +			+ + + + +										
160	+ + + + P	– – – – –					T T – – –							
163	– – – – –	– – – – –		+ + + + +										
168	+ + + + +	+ + + + +												
170	– – – – –			+ + + + +										
185	– – – – –			– – – – –				T – – –						
187	+ + + + +		+ + + + +											
191	– – – – –	+ + + + +		+ + + + +		P T – – –	+ + + P T		+ + + + +					+ + P T –
194	– – – – –			+ + + + P			+ + P P T							
201	+ + + + +	+ + + + +												
204	+ + + P T	+ + + + +												
214	+ + – – –	– – – – –		– – – – –		– – – – –	T – – – –			P P T – –		P T – – –	T – – – –	
219	– – – – –	– – – – –		P T – – –		– – – – –	T – – – –			P T – – –				
222	+ + + + +			+ P T – –			+ + P T –			+ P T – –				
224	– – – – –			+ + + P T								P P T – –		
225	– – – – –	+ + + P –		– – – – –										
227	+ + + P –	+ + + P –					+ + P T –					+ + P T –		
229	– – – – –	+ + P – –		+ + + + +										
230	– – – – –	+ + + + +		+ + + + +			+ + + + +							
232	– – – – –			+ T – – –		– – – – –	T – – – –		P T – – –	T – – – –			T – – – –	
237	+ + + + +			+ + + + +										
239	+ + + + +	+ + + + +		+ + + + P		+ + + + P								
410	– – – – –	– – – – –		P T – – –			T – – – –							
411	+ + + + +			+ + + + +			+ + + P P							
412	– – – – –	+ + + + +	+ + + + P	+ P T – –			+ + P P T				+ P T – –			P T – – –
414	– – – – –	+ + + P P		+ + + + +				+ P P T –		P P T – –	– – – – –	– – – – –		– – – – –
415	– – – – –			– – – – –			– – – – –			P T – – –				
418	– – – – –			– – – – –			P T – – –			+ P T – –			P T – – –	
419	– – – – –	+ + + + +		P T – – –			P T – – –			P T – – –				
421	+ + + + +		+ + + + +	+ + + + P			+ + + + P							
29	+ + + + +			+ + + + +			+ + + + +							
30	+ + + + +			+ + P T –										
7	+ + – – –			+ + + + +		+ + + + +								+ + + + +
4	+ + + – –	+ + + – –		+ + + P T			P T – – –			+ P P – –	P P T – –			+ + + + +
5	– – – – –	+ P – – –		+ T – – –						+ + P T –		+ + P T –		+ + P – –
56	+ + + + +	+ + + + +		+ + + + +										
65	+ + + + –	+ P – – –		+ + + P T			+ + P T –					+ + P T –		+ + P – –
66	– – – – –	+ + P – –		+ + + P P			+ + + + P							
181	+ + + + +			+ + + + +			+ + + + P							
182	+ + + + +	– – – – –		+ + + + +										
209	– – – – –	P – – – –												

TABLE Ia—*Trend of Brucella agglutination reactions in cattle over a seven-year period*

TABLE Ia—*Trend of Brucella agglutination reactions in cattle over a seven-year period*

Animal	5-24	6-25	6-27	2-28	11-28	2-29	4-29	7-29	9-29	11-29	2-30	4-30	5-30
1	+ + – – –		– – – – –			+ + P T –							
2	– – – – –		+ + T T –			+ + P P T							
8	– – – – –		+ + P – –			P P T – –			+ P T – –	P P T – –		P T – – –	+ P T – –
10	– – – – –		– – – – –			T – – – –			– – – – –		– – – – –		
54	– – – – –	– – – – –	+ + + P T				+ + + + +						
98	– – – – –		– – – – –			P T – – –		+ P T – –	+ + P – –	+ P T – –			
104	– – – – –		– – – – –			+ + P T –			+ P P T –	+ + P T –			
105	– – – – –		+ + + + +	+ + + P P		+ + + + +							+ + + + P
107	– – – – –		+ + P P P							+ + P T –			
110	– – – – –		+ + P – –			+ + P T –			+ + + P T	+ + + + P			
216	+ + + + +		+ P T – –										
257	+ + + + +		+ + + + +			+ + + + +							
258	P – – – –		– – – – –			+ + + + +					+ + + + +		
270	+ + + + +		+ P T – –										
277	– – – – –		+ + + + +		+ + + P T	+ + + P P							
282	+ + + + +		– – – – –			P T – – –							
297	+ + + + +				+ + + + P			+ + + + +					
298	+ + + + +		+ + + + +			+ P P T –							
299	– – – – –		– – – – –			T T – – –							
300	+ P – – –					T – – – –							
301	– – – – –		– – – – –			T – – – –		– – – – –	P T – – –				
307	+ P – – –		T – – – –										
309	+ + + + +		+ + + + +			+ + + + +			+ + + + +				
426	– – – – –		P P T – –			P T – – –			P T – – –		T – – – –		
428	– – – – –		– – – – –			– – – – –			T – – – –		– – – – –		
192	– – – – –	– – – – –	– – – – –			– – – – –		T – – – –	– – – – –			– – – – –	
196	– – – – –		+ T – – –			+ + P P T					+ + P T –		+ + + T –
413	– – – – –	+ + + + +	+ + + + +			– – – – –			+ + P T –	– – – – –			+ + + + +

TABLE Ib—*Trend of Brucella agglutination reactions over a six-year period*

Animal	6-25	6-27	2-28	11-28	2-29	4-29	7-29	9-29	11-29	2-30	4-30	5-30
9	– – – – –	+ + + + +			+ + + P P		+ + + + +					
12	– – – – –	– – – – –			P T – – –							
99	– – – – –	+ + + + +			+ + + + +		+ + + + +					
108	– – – – –	+ + + + +			+ + P T –		P P T – –	+ P T – –				+ + P T –
115	– – – – –	– – – – –		– – – – –	– – – – –		T – – – –	– – – – –			– – – – –	– – – – –
283	+ + + + P			P P P – –	+ + P T T							
303	+ + + + +	+ + + + P										
305	+ + + + +	+ + + + +		+ + + + P								
320	+ + + + P	+ + + + +			+ + + + +							
424	– – – – –	P T – – –			T – – – –		+ P T – –	+ P P T –	T – – – –	T – – – –		
427	– – – – –	+ + + + +										
429	– – – – –	+ + + + +			– – – – –		– – – – –	– – – – –			– – – – –	
430	– – – – –	+ + + + +			P T – – –			+ + + + +			+ + + + P	
288	+ + + + +				+ + + + +							
319	+ + P – –	– – – – –		– – – – –	T – – – –				– – – – –		T – – – –	

TABLE Ic—*Trend of Brucella agglutination reactions over a four-year period*

Animal	6–27	2–28	11–28	2–29	4–29	7–29	9–29	11–29	2–30	4–30	5–30
3	P – – – –			– – – – –							
13	+ + + + P			+ + + + +					+ + + + +		
14	+ + + + P			+ + + + +	+ + + + +						
15	+ + + + +			+ + + P T						+ P T T –	+ + + P –
17	– – – – –			P T – – –	P P T – –		+ P T – –	P T – – –			
18	+ P T – –		T – – – –	T – – – –			T – – – –			T – – – –	
19	+ + + P P			+ + P T –		+ P T – –	+ + P T –	+ + P T –			
22	– – – – –			+ + + + +		+ + + + +					+ + + + +
23	+ + + P T									+ P T – –	– – – – –
26	+ + + + P							+ + + + +	+ + + + +		
60	– – – – –			T – – – –			T – – – –		P T – – –		
97	+ + + + +		+ + + P P			+ + + + +					
112	+ + + + P		+ + + P P								
113	+ + + + +			+ + + + P							
114	+ P P – –			+ P T – –		+ + P T –	+ P T – –	+ + P T –			
116	+ + + + +						+ + + + +				
117	+ P T – –			+ + + + +							
122	+ + + + +		P T – – –	+ + P P T				+ + + P T			
123	+ + + + +						+ + + + +				
127	P – – – –			+ + P T –			+ + + P T				
128	+ + + + +			+ + + + P							
129	– – – – –		T – – – –	– – – – –			P T – – –		P T – – –	P T – – –	
130	– – – – –			+ + P T –							
131	– – – – –			T – – – –			– – – – –				
132	+ + + + +			+ + + + +		+ + + + +					
133	+ + P – –			+ P P T –	P P T – –	+ + + + +			+ P T – –		+ + P – –
134	+ + P – –			+ + + + +							+ + + + +
135	+ + + + +			+ + + + +							
136	+ + P T –			+ + + + +							
138	+ + + + +			+ + + + +							
141	+ + + + +		+ + + + +								
143	+ + + + +				+ + + + +						
146	+ + + + T			P P T – –							
154	+ P P T –		+ + + P P	+ + + + P				+ + + P T			
157	+ + P – –			+ P T – –			+ P T – –				
161	– – – – –			P P T – –							
162	– – – – –			+ + + + +							
167	– – – – –			+ + + + +							
172	+ + + + +				+ + P T –		+ + + P T				
178	+ + + + +			+ P P T –					+ + + P T		
187	– – – – –		– – – – –	– – – – –			– – – – –		+ + P T –		+ + + + P
193	– – – – –			– – – – –		T – – – –	T – – – –			T – – – –	
234	– – – – –			+ + + + +		+ + + + +					
236	+ + + + +		+ + + + +	+ + + + +							
247	+ + + + +			+ + + + +		+ + + + +					+ + P T –
249	+ + + + +			+ + + + +	+ + + + +						

TABLE Ic—*Trend of Brucella agglutination reactions over a four-year period—Continued*

Animal	6–27	2–28	11–28	2–29	4–29	7–29	9–29	11–29	2–30	4–30	5–30
255	T – – – –			+ + + + +			P T – – –				
259	– – – – –		– – – – –	T – – – –					+ + + + +		
266	+ + + + +			P T T – –			P T – – –	– – – – –	+ P P T –		– – – – –
269	+ + + P P			T – – – –				+ + P – –			
279	P – – – –			+ + + + +							
288	+ + + + +			P P T – –							
290	+ + + P P		– – – – –	+ + + + +							
293	+ + + + +			+ + + + +							
294	+ + + + +			+ P T – –	P P T – –		P T – – –				P P T – –
295	+ + P – –		P T – – –	P P T – –			P T – – –	P T – – –		P T – – –	– – – – –
302	P P – – –							T – – – –			
306	– – – – –			+ + P T T							
310	+ + P P –			P T T – –		+ + P T –					+ + + + +
311	+ T – – –			– – – – –		+ + + + +					
312	– – – – –		+ + P T –	+ + + P P	+ + + + +						
314	– – – – –		– – – – –	– – – – –		T – – – –	– – – – –		– – – – –		
322	– – – – –			+ + + + +					+ + + + +		
326	– – – – –			+ + + + +					T – – – –		– – – – –
328	– – – – –			T – – – –		– – – – –	P – – – –		P P T – –		
330	– – – – –			– – – – –		– – – – –	+ P T – –	P T – – –		P P T – –	
331	– – – – –		+ P T – –	+ + P T –	P P T – –	P T – – –	P T – – –				
333	– – – – –		T – – – –	+ P T – –	P T – – –			+ + + + +			
337	– – – – –			– – – – –							
338	+ + + + P		– – – – –	T – – – –							
340	– – – – –		P P T – –	+ + + P T							
342	– – – – –			+ + + + +							
344	+ P – – –		– – – – –	+ + + P P							
345	– – – – –		P T – – –	P T – – –		– – – – –	T – – – –				
347	– – – – –			+ + P T –							
401	– – – – –			+ + P P P							
416	+ + + + +			T – – – –			T – – – –				
417	+ + + + +			+ + + P T							
422	– – – – –			T – – – –			P T – – –				– – – – –
423	+ + + + +			T – – – –		+ + P T –	+ P T T –	– – – – –			
425	T – – – –			– – – – –							
431	+ + + + P			+ + + P P							
432	+ + + + +		T – – – –	T – – – –							
195	– – – – –			+ + + + P							
433	+ + + + +			+ + + P P							
435	+ + + + P			+ + + + P		+ + + + +					
436	+ + + + P			T – – – –		– – – – –	– – – – –		– – – – –		– – – – –
437	– – – – –			+ + + + P							
438	– – – – –			T – – – –			T – – – –		P P T – –		
439	– – – – –			P T T – –			+ P T – –			+ P T – –	– – – – –
440	– – – – –										

TABLE Id—*Trend of Brucella agglutination reactions over a three-year period*

ANIMAL	2–28	11–28	2–29	4–29	7–29	9–29	11–29	2–30	4–30	5–30
268	– – – – –				T – – – –		+ + P T –			– – – – –
267	– – – – –				T – – – –		– – – – –			
31		– – – – –			T – – – –	T – – – –	T – – – –	– – – – –		
43		– – – – –	– – – – –			T – – – –		T – – – –		
45		T – – – –			T – – – –	T – – – –		– – – – –		
46		– – – – –	T – – – –						T – – – –	
47		– – – – –	T – – – –		+ P T – –	P T – – –	+ P T – –			– – – – –
49		– – – – –			– – – – –	T – – – –	– – – – –	– – – – –		
51		T – – – –	T – – – –			T – – – –		– – – – –		
52		T – – – –			– – – – –		P T – – –			
58		– – – – –			T – – – –		T – – – –	T – – – –		
139		P T T – –	P T – – –							
151		P T – – –	+ + P – –			T – – – –		T – – – –	– – – – –	
153		– – – – –	– – – – –			P P T – –	P P T – –			– – – – –
169		P T – – –	+ + P – –		+ + P T –	+ P T – –	T – – – –			P T – – –
171		T – – – –	P T – – –							
173		P P T – –	P T – – –			– – – – –			P T – – –	
184		P T – – –	+ P T – –	P T – – –			P T – – –			
190		P P T – –	P P T – –							
207		– – – – –	– – – – –	P T – – –						– – – – –
216		T – – – –	– – – – –	T – – – –		– – – – –				
263		+ + + + P				+ + + + +				
273		T – – – –			P T – – –		T – – – –	P T – – –		
274		– – – – –	T – – – –		T – – – –	– – – – –			– – – – –	
276		– – – – –				P T – – –				
284		– – – – –			T – – – –		– – – – –		T – – – –	
285		T – – – –	T – – – –			P T – – –				
291		– – – – –		P T – – –	– – – – –	T – – – –		T – – – –		
292		– – – – –	– – – – –	P P T – –	T T – – –			– – – – –	– – – – –	
296		– – – – –					P T – – –	– – – – –		
304		– – – – –		T – – – –			– – – – –	– – – – –		
313		– – – – –					T – – – –	– – – – –		
316		– – – – –		– – – – –	T – – – –			– – – – –		
318		– – – – –			T – – – –		P T – – –	– – – – –		
321		P T – – –	+ + + P T		+ + + P T		T – – – –		P T – – –	
324		– – – – –					– – – – –			
327		– – – – –			T – – – –					

TABLE Id—*Trend of Brucella agglutination reactions over a three-year period—Concluded*

ANIMAL	2-28	11-28	2-29	4-29	7-29	9-29	11-29	2-30	4-30	5-30
335		– – – – –			T – – – –		– – – – –		T – – – –	
350		– – – – –	T – – – –			P T – – –				T – – – –
352		– – – – –	T – – – –	– – – – –		– – – – –			– – – – –	
357		T – – – –	P T – – –				+ P T – –			– – – – –
360		– – – – –	T – – – –							
363		– – – – –	T – – – –	P T – – –						
364		– – – – –	T – – – –	T – – – –		T – – – –		T – – – –		T – – – –
366		T – – – –	P T – – –			– – – – –		P T – – –		
369		– – – – –			– – – – –	– – – – –	T – – – –	– – – – –		
372		T – – – –	P T – – –			+ P T – –				
374		– – – – –	T – – – –			– – – – –		– – – – –	T – – – –	
377		– – – – –	T – – – –			T – – – –				
378		– – – – –	T – – – –							
379		– – – – –	T – – – –			P T – – –	– – – – –	T – – – –		
382		+ + + P P	+ + + P P				+ + + + P			
383		– – – – –			T – – – –		– – – – –		– – – – –	
384		– – – – –	– – – – –			T – – – –				– – – – –
391		– – – – –		T – – – –						
408		+ + + P T				+ + + + +				
448		– – – – –	– – – – –			P T – – –			– – – – –	
450		– – – – –	– – – – –				T – – – –			
451		– – – – –			T – – – –		P T – – –	– – – – –		
452		– – – – –	– – – – –			T – – – –		– – – – –	– – – – –	
453		T – – – –	– – – – –	P P T – –		P – – – –				– – – – –
454		– – – – –		T – – – –	P T – – –	+ + P – –	– – – – –		T – – – –	T – – – –
455		– – – – –		+ + P T T		+ P P T –	– – – – –	P T – – –		
456		– – – – –		– – – – –		T – – – –		– – – – –		
140		P T T – –	P T – – –							
243		+ P T – –	P P T – –			+ + P T –	+ + + P T			
253		– – – – –			– – – – –	T – – – –				– – – – –
260		– – – – –			T – – – –	T – – – –	– – – – –	T – – – –		
264		– – – – –			T – – – –		– – – – –	– – – – –		
272		– – – – –	T – – – –			– – – – –				– – – – –
289		– – – – –			– – – – –		T – – – –			– – – – –
479		– – – – –								T – – – –
487		+ P – – –			P T – – –	P T T – –		– – – – –		

TABLE Ie—*Trend of Brucella agglutination reactions over a two-year period*

Animal	2–29	4–29	7–29	9–29	11–29	2–30	4–30	5–30
41	T – – – –				T – – – –	+ + + + +		
137	– – – – –					– – – – –		T – – – –
152	P T – – –			P T – – –		– – – – –		– – – – –
166	T – – – –		– – – – –	– – – – –				
179	+ + P – –				+ + P T –	– – – – –		– – – – –
180	T – – – –		– – – – –	– – – – –				
188	+ + + P T	+ + P T –					– – – – –	
197	T – – – –				T – – – –	+ P T – –		– – – – –
203	– – – – –							+ + + + +
205	T – – – –			T – – – –		– – – – –		
206	– – – – –			P T – – –	T – – – –			
339	– – – – –							+ + + + +
346	+ + + P P			+ + + + +				
348	+ + + + +			– – – – –			– – – – –	
349	T – – – –			T – – – –	– – – – –	– – – – –		
354	– – – – –			P T – – –	+ P T – –			
356	P T – – –			P T – – –	+ P T – –			P T – – –
361	P T – – –						P T – – –	
362	P T – – –		– – – – –	– – – – –				+ + + + +
365	+ + + + +			– – – – –				
368	T – – – –					+ + + + +		
370	+ + + + +				+ + + + +	+ + + + +	+ + + + P	
375	– – – – –					P T – – –		T – – – –
376	T T – – –			– – – – –		– – – – –		
380	– – – – –			P T – – –		+ + + + +		– – – – –
381	T – – – –							T – – – –
385	+ + + + +			T – – – –		– – – – –		
387	– – – – –			T – – – –		P T – – –		
388	T – – – –						T – – – –	
389	P P T – –			– – – – –				
393	T – – – –				– – – – –			
394	– – – – –							

TABLE Ie—*Trend of Brucella agglutination reactions over a two-year period—Continued*

Animal	2–29	4–29	7–29	9–29	11–29	2–30	4–30	5–30
402	P T – – –			– – – – –	– – – – –	– – – – –		
403	+ + + + +					+ + + + P		
404	+ + + + +	+ + + + +						
443	P T – – –			P T – – –		T – – – –		
447	– – – – –			T – – – –		– – – – –		
458	+ + P T –			P P P T –	+ + + + +			
459	– – – – –			P T – – –	P T – – –	– – – – –		
460	T – – – –			+ + P T –			P T – – –	
275	+ + + + P			+ + + + +				
315	+ + + + +	+ + + + +						
461	+ + + + P						P T – – –	+ + P – –
462	P P T – –			+ P P T –	P T – – –			
463	+ + P – –				+ P T – –			+ P T – –
464	+ + P P T			+ + P T –	+ + P T –		+ + P T –	+ + + T –
465	T – – – –			T – – – –		– – – – –		
467	+ + + + +			+ + + + +				
468	+ + + + +	+ + + + +						+ + + P T
469	+ P T T –	+ + P T –						
470	+ + P P T	T – – – –						+ + + + +
471	P T T – –			+ + + + +				
472	+ + + + +			+ + + + +				
473	+ + + P T					T – – – –		+ P – – –
474	T – – – –		– – – – –	T – – – –				
475	T – – – –	P T – – –		T – – – –		T – – – –		– – – – –
476	T – – – –				– – – – –		– – – – –	
477	T – – – –				P T – – –	– – – – –		
478	P P T – –			+ + P T –				
482	P T – – –			– – – – –				– – – – –
483	+ + + + P					+ + + P T		
484	– – – – –		+ + + + P			T – – – –		
485	T – – – –			+ + + + +				
486	T – – –			+ P T – –	P T – – –			
	P							

TABLE Ie—*Trend of Brucella agglutination reactions over a two-year period—Concluded*

Animal	2–29	4–29	7–29	9–29	11–29	2–30	4–30	5–30
176		T T – – –		T – – – –		– – – – –		
177		T – – – –		T – – – –		– – – – –		
213		P T – – –		P T – – –		P T – – –		T – – – –
220		+ P T – –		P T – – –		T – – – –		T – – – –
226		P T – – –						– – – – –
325		P T T – –		P P T – –		+ P T – –		
359		T – – – –		+ P T – –	P T – – –			
400		P T T – –						– – – – –
489		+ + + + +						P T – – –
210		T – – – –		P T – – –		– – – – –		
211		P T – – –		P T – – –		– – – – –		
399		– – – – –						P T – – –
164		+ P T – –			P T – – –			
250				– – – – –	– – – – –	T – – – –		– – – – –
254				– – – – –		T – – – –		
265				+ + P – –	+ + P T –			
351				P T – – –	– – – – –	T – – – –		
142				P T – – –		P T – – –	T – – – –	– – – – –
147				T – – – –	T – – – –	– – – – –		– – – – –
150				T – – – –	P T – – –			
155				T – – – –		T – – – –		
159				P T – – –		+ + + + +	+ + + + +	P P T T –
186				– – – – –	– – – – –			
228				+ + + + +	+ + + + +		– – – – –	
233				T – – – –				+ + + + +
235				+ + + + +				
240				T – – – –	T – – – –	– – – – –		
245				T – – – –		T – – – –	+ P T – –	– – – – –
252				– – – – –	T – – – –	– – – – –		
396				T – – – –	– – – – –		– – – – –	
174					T – – – –		T – – – –	
144					T – – – –	– – – – –	– – – – –	

expulsion of the fetus, she was promptly isolated from the herd. Heifers were maintained either in barns or on farms separate from the older animals. All parturitions took place in a maternity barn maintained solely for that purpose.

An analysis of the reactions set forth in table I shows that those animals which gave complete agglutination reactions in the 1:100, 1:200 and 1:500 dilutions, on one or more tests, constitute 34.9 per cent of the total number of animals in the herd during the eight test-years.

In table II is set forth the percentage of animals that, having once given a complete agglutination reaction in one of the titres used, have reverted to a trace, incomplete or negative reaction. These percentages are based on two or more consecutive tests. Those showing this drop in titre on only one test and where no succeeding tests were made, or if the negative or incomplete reaction later showed a complete agglutination in the 1:25 dilution or above, were not included in these percentages.

From these figures it would appear that only a small percentage of animals reacting in a titre of 1:100 or above become permanently negative. A significant percentage of those showing reactions below 1:100 revert to a point sufficient to class them as negative reactors. It is of interest to note, however, that most all animals that react to the agglutination test, even though they

TABLE If—*Trend of Brucella agglutination reactions over a one-year period*

ANIMAL	5–30	6–30
196	+ + + + +	+ + + + +
208	+ + P T −	+ + + + +

TABLE II—*Summary of reacting animals reverting to incomplete and negative reactions*

		AGGLUTINATION TITRE			
		+ 1:25	+ 1:50	+ 1:100	+ 1:200 AND 1:500
Total Number		26	32	24	165
Reverting to Partial or Trace in 1:25	No.	8	10	3	9
	%	30.8	31.2	12.5	5.5
Reverting to Negative	No.	0	0	2	2
	%	0	0	8.3	1.2

do not remain infected, retain a trace of agglutinins in their blood serum for many years.

Of the 165 animals showing a high agglutination titre (1:200 and above), 35 (21.2 per cent) showed a significant continuous decline in titre. Of the 24 showing a reaction no higher than 1:100, 11 (45 per cent) also showed a marked decline in titre. On successive tests the declining titres, as a rule, tended to become lower rather than higher. It is difficult to interpret the decline in titre of this number of animals in the absence of complete bacteriological data, especially on the milk. If the decrease in titre of such a percentage of high-reacting animals indicates recovery from infection, this should be considered when the question arises as to how they should be disposed of. In other words, if these figures are of any significance, will such a method of eradicating the disease as that of slaughter ever be a logical one to pursue where valuable animals are involved?

The number of abortions for the different classes of reactors, including those that have always been negative for the total number of test-years that they were in the herd, are set forth in table III. The differences in the number of abortions, based on percentages, furnish an interesting comparison of the abortion rate between those that have always remained negative to the agglutination test and those that have shown a complete agglutination in one or more of the five serum dilutions employed.

These percentages, of course, do not furnish any information on the yearly abortion rate of the different classes of reactors but they do, however, furnish information on the average abortion incidence of reacting animals and those that remain negative over a long period of time.

TABLE III—*Number of abortions occurring in animals during the period of observation and summarized under their maximum agglutination titres*

		Agglutination Reaction					
		Negative	Trace and Incomplete in 1:25	Complete in 1:25	Complete in 1:50	Complete in 1:100	Complete in 1:200 and 1:500
Total No. of Animals		143	151	26	32	24	165
Aborting	No.	22	39	6	12	9	88
	%	15.4	25.8	23	37.5	37.5	53.3

As one would expect, in the high-titre reactors are to be found the largest number of animals that abort. Although only 88 animals in this class aborted, they were actually responsible for 136 abortions. Many of these animals aborted two or three times. Two of them aborted four times. The number of abortions per animal was much lower for those reacting below 1:200 and those that have always remained negative. In the 1:100 class, 9 animals were responsible for 13 abortions; in the 1:50 class, 12 animals were responsible for 15 abortions; in the 1:25 class only 6 abortions occurred; in the trace and incomplete class, 39 animals were responsible for 42 abortions; in the negative group, 25 abortions occurred among the 22 aborting animals.

The difference of 10 per cent in the abortion rate between the negative animals and those that showed a trace or incomplete reaction in a 1:25 dilution is difficult to explain if the abortions in the latter group are not attributed to Brucella. It is interesting to note that when these two groups are added, the abortion rate is 20.5 per cent, a figure that has been given as the approximate percentage of abortions not due to Brucella.

The differences in the abortion rate of each class of reactors cannot be explained on the basis of a difference in the length of time the animals were under observation in this herd. The per-

TABLE IV—*Number of test-years animals were in herd*

Number of animals showing:		Test-Years in Herd							
		1	2	3	4	5	6	7	8
Trace and Incomplete in 1:25	No.	5	64	49	11	1	3	13	5
	%	3.31	42.38	32.45	7.28	0.66	1.98	8.60	3.31
Complete in 1:25	No.	5	6	7	3	0	2	1	2
	%	19.23	23.07	26.92	11.53	0	7.69	3.84	7.69
Complete in 1:50	No.	4	3	10	2	0	6	4	3
	%	12.50	9.34	31.25	6.25	0	18.75	12.50	9.34
Complete in 1:100	No.	1	4	5	2	1	0	6	5
	%	4.16	16.66	20.83	8.33	4.16	0	25.0	20.83
Complete in 1:200 and 1:500	No.	13	24	38	18	15	12	29	16
	%	7.87	14.60	23.03	10.90	9.09	7.27	17.63	9.69

centage of animals, in the five classes of reactors, as is illustrated in table IV, that were in the herd for at least four test-years, do not differ widely. Further, with the exception of the 1:25 class there is not a great difference in the percentages for each class of reactors that were present in the herd for 6, 7 and 8 test-years.

The material presented in this paper should be considered as only accumulated data on the trend of the Brucella agglutination titre in cattle and the abortion rate for different classes of reactors and negative animals over a period of several years. We realize that the figures presented may not obtain for animals in small herds or a similar number of animals in a herd maintained under different conditions. Not every herd, as this one was, is under the constant care and supervision of a veterinarian, nor is the same practice carried out as regards replacement of animals. There are herds in which most of the replacements are raised from calfhood and there are those in which a practice is made of purchasing older animals from other herds for replacement stock. In this particular herd both replacement procedures have been and are being practiced.

It is hoped that these data will stimulate others to present similar data on herds that have been under close supervision for several years. Such data will reveal more clearly what occurs in the natural course of Brucella infection in cattle.

Cattle-Dipping Season Opened

Another active tick-eradication season opened the latter part of March and the extension of the campaign to new areas in the South was announced by veterinary officials of the U. S. Department of Agriculture, which is coöperating with the state and local officials in a well-organized campaign against this fever-bearing parasite. During the winter months active preparations had been in progress in many sections of the South. The advance of the eradication campaign had been heralded and explained by motion pictures, publications, and instructive talks. Hundreds of dipping vats were constructed and thousands of gallons of arsenical dip were on hand for preparing the tick-destroying bath. Forces of trained inspectors were assembled to direct the systematic cattle-dipping campaign, at strategic points in Texas, Florida and Arkansas. Louisiana continues to mark time in this project, as the law is inoperative due to lack of funds. These four states contain the only areas still under federal quarantine because of the presence of cattle ticks.

COOPERATION OF COUNTY AGENT AND VETERINARIAN*

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The word cooperation is one very often heard in our day. A good definition of the word is "working together," which implies more than one person. We, therefore, want to say something about county agents and veterinarians working together.

One of the duties of a county agent is to get folks to work together. This is so important that the success of his program depends very largely upon his ability to get folks to work together. Some one has said that the way to have friends is to be friendly. One might say also that the way to get cooperation is by cooperating. There are lots of folks who cooperate just half way. They are willing to do the cooing and let the other fellow do the operating.

The duties of a county agent are many and varied. He is supposed to know something about most everything pertaining to farm life. Many times he is supposed to know much more than it is humanly possible for one man to know. If he is wise, he will never give advice beyond his actual knowledge.

This is a day of specialization and no man can specialize in many subjects. A county agent, to do his work well, must of necessity know something of the laws of sanitation affecting profitable live stock production, but he is not supposed to be a practicing veterinarian, unless he has been graduated in veterinary medicine. A county agent should know enough about modern veterinary methods to be able to advise his farmers against splitting cows' tails for "wolf in the tail" and sawing off the horns for "hollow horn." He should be well enough informed to be able to warn against many uses and practices often found among some farmers. There are some remedies used among the more ignorant that are worse than the disease. A county agent should know enough about the ordinary diseases of animals to diagnose them partially and advise the services of a veterinarian. This service will often save a serious outbreak of disease and financial loss to the farmers.

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The county agent does not pose as a specialist in any line. The colleges of agriculture have specialists on various subjects who visit the various counties and offer their help. The local veterinarians are looked to as the specialists in animal diseases, and fortunate is the county agent who has the veterinarian to rely on for doing this specialized work. The county agent has more work to do than he can ever get done, without hunting any extra job. His work is largely in giving advice and demonstrating modern agricultural practices.

A part of each county agent's program is devoted to getting better animals on the farm. As the standard of live stock is raised, the aid of the veterinarian is more in demand. Scrub stock of any kind rarely ever receives medical attention. It is the pure-bred animal whose life is valuable and for which the owner is willing to pay for expert help. The county agent who is really putting over a worthwhile job is creating at the same time more work for the veterinarian.

There is really no reason for conflict or duplication in the work of county agent and veterinarian. Each should create demand for the services of the other. The line between the services of the two is clear and distinct. There may be instances when some advice from the county agent might prevent the calling of a veterinarian, while on the other hand there are probably more instances when his advice would call for the veterinarian. The same may be true with reference to the doctor for the family. There are certain rules for prevention of disease among folks which a county agent should know and to which he should call attention whenever the violation of these rules comes under his observation. But it would be a foolish county agent who would attempt to take the place of the physician.

In building the live stock industry the health of the animals must always be of first consideration, and to this phase of the work the veterinarian must continue to be relied upon. County agency work was never intended to displace the services of the veterinarian, but is and should be a means of creating more work for the veterinary profession by creating a better grade of live stock whose lives are worth saving. Ours is the work of building and yours is the work of conserving and saving. If this is the true interpretation of the work of county agent and veterinarian, there should always be cooperation, each with the other, building together a better live stock industry for our country.

RESULTS OF EXPERIMENTS WITH THE USE OF PIGEON-POX VIRUS AS CUTANEOUS VACCINE AGAINST FOWL-POX*

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Fowl-pox has been recognized as one of the most disagreeable diseases with which the poultryman has to deal. This fact is due, not only to its highly contagious nature, which makes it possible to spread rapidly in a flock as well as to the flocks in a community, but also to the possibility of its appearing on the same place from year to year after once having been introduced.

Various treatments usually suffice to bring about a cure to affected individuals, but these do not solve the problem, since they do not check the spreading of the disease.

Vaccination for the prevention of this disease has been practiced for a number of years, by using the fowl-pox virus in various ways as a vaccine for this purpose. While some of these methods have given fairly good results when used as recommended, none have been entirely without objection, since in most cases the living virus has been used and in others attenuation of it has been tried in various ways.

Where the living fowl-pox virus has been used as vaccine for this disease, there has usually been noticed a marked reaction in the bird, sometimes not much different from the disease itself. Usually vaccination by the living virus is followed by a decrease in egg-production when administered to laying stock.[1] The greatest objection, however, is that the virus is introduced on the place and remains a potential danger at all times for susceptible birds. The various methods of attenuating this virus have not been satisfactory, as the product usually is dead at the time it is used, and therefore no immunity is produced, or else none different from the live vaccine.

In recent years considerable work has been done by several investigators in this country, as well as in European countries, in an attempt to find a more satisfactory immunizing product for this disease.

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In 1927, Dr. deBlieck, of Holland, reported at the World's Poultry Congress held at Ottawa, Canada, on a product which was called anti-diphtherin, which was giving practically perfect results in that country as a vaccine against fowl-pox. In this report no information regarding the preparation of this product was given. This paper did, however, stimulate further research in a vaccine for this disease, both in this country and abroad.

Much has been learned in recent years concerning the relationship of different filtrable viruses, and use of this knowledge has

FIG. 1. Preparation of the breast of pigeon for application of the virus to the open feather follicles.

been made in the preparation of vaccines for various diseases. The oldest example of this is the use of cow-pox virus as an immunizing agent against smallpox in the human family.

In 1929, Zwick et al,[2] tried attenuating fowl-pox virus by repeatedly passing it through pigeons and using this attenuated virus as vaccine. Their report indicates that strong immunity results following its use.

In 1930, Doyle[3] reported on the use of pigeon-pox virus as a vaccine against fowl-pox. His report indicates that it is very

satisfactory as an immunizing agent against natural infection as it occurs in England.

Preparation of Vaccine

In making vaccine from chicken-pox virus, the massive crusts or scabs on the combs of artificially infected Leghorn cockerels are usually collected 15 or 20 days following inoculation and dried in a desiccator. These dried scabs are then ground into powder and stored at a low temperature until use, when this

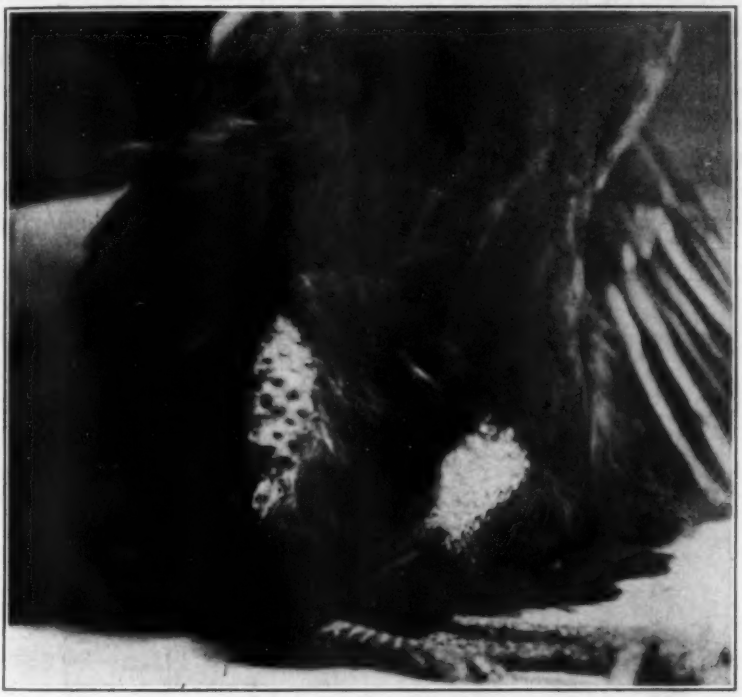

Fig. 2. Feather follicles eight to ten days after the application of the virus to the anterior tibial region.

powdered virus is either suspended in distilled water or dilute glycerin.

To grow pigeon-pox virus, we cannot follow this method, as the pigeon does not have a large comb on which to grow it, and because the growth of pigeon-pox virus produces a more moist scab, and also involves the deeper tissues of the skin.

Following the suggestions by English workers, it was learned that this virus would grow abundantly on the breasts of pigeons when applied to the open feather follicles. This condition is illustrated in figure 1.

After the application of the virus, scabs usually develop sufficiently in 12 to 14 days. These scabs are then removed either with a scalpel or a small curette, and collected in a tared Petri dish. This pulp is then ground in a mortar with the addition of ten parts of diluted glycerin, consisting of equal amounts of glycerin and distilled water. After grinding, this material is filtered through gauze and the vaccine is stored in a refrigerator until it is used.

Fresh virus prepared as described above was used to vaccinate a pen of 74 pullets that were just coming into production. The follicle method of vaccination was used, which consists of pulling out 8 or 10 feathers in the anterior tibial region, near the tibiometatarsal joint, and applying a small amount of the vaccine to the feather follicles by the use of a brush. Eight to ten days following the application of the vaccine, the feather follicles become swollen, as shown in figure 2. Seventy-two of these birds

TABLE I—*Pen 1 (74 pullets)*

BEFORE VACCINATION		AFTER VACCINATION	
DATE	EGGS	DATE	EGGS
Nov. 1, 1930	4	Nov. 18, 1930*	9
2	5	19	8
3	8	20	11
4	6	21	13
5	9	22	8
6	5	23	14
7	10	24	15
8	9	25	7
9	13	26	12
10	8	27	15
11	10	28	16
12	7	29	13
13	15	30	9
14	9	Dec. 1	14
15	12	2	15
16	8	3	17
17	11	4	10
		5	13
		6	17
		7	12
		8	18
		9	14
		10	19
		11	13
		12	17
		13	18
		14	16
Totals	149		363
Average per day	8.7		13.9

*Vaccinated.

had perfect "takes." The other two were revaccinated and "takes" resulted, indicating that possibly not enough vaccine was rubbed into the feather follicles on these two the first time.

The pigeon-pox virus used in our experiments was a pure strain obtained from England through the United States Bureau of Animal Industry.

Records of egg-production were kept three weeks before vaccination and four weeks following (table I).

A cockerel, artificially infected with chicken-pox virus, was placed in pen 1, on December 13, the 25th day following vaccination of this pen of birds with pigeon-pox virus vaccine. This cockerel had extensive lesions on comb and wattles, which were picked off and eaten by the vaccinated pullets, but none developed symptoms or lesions of the disease.

TABLE II—*Pen 3 (87 pullets)*

BEFORE VACCINATION		AFTER VACCINATION	
DATE	EGGS	DATE	EGGS
Nov. 14, 1930	4	Dec. 2, 1930*	8
15	6	3	9
16	7	4	10
17	3	5	12
18	8	6	9
19	10	7	10
20	9	8	13
21	9	9	14
22	9	10	6
23	7	11	11
24	10	12	13
25	14	13	10
26	11	14	12
27	10	15	9
28	9	16	14
29	7	17	13
30	8	18	16
Dec. 1	11	19	8
		20	12
		21	16
		22	19
		23	21
		24	15
		25	14
		26	13
		27	18
		28	19
		29	21
		30	20
Totals	154		385
Average per day	8.5		13.7

*Vaccinated.

Pigeon-pox virus vaccine, prepared according to the method described by Doyle,[3] was used to vaccinate pen 2, consisting of 65 cockerels, on November 24.

On December 20, or nearly one month following vaccination, a cockerel with heavy chicken-pox lesions on comb and wattles, from artificial infection, was placed in this pen. No cases of the disease have developed, nor have the birds shown any ill effects.

On December 2, pen 3, consisting of 87 pullets, was vaccinated with pigeon-pox virus vaccine prepared as for pen 1. Records of egg-production were kept as for pen 1, with the results shown in Table II.

The general condition of the birds was at no time affected.

Six cockerels from pen 2 were artificially infected on December 15, or three weeks following vaccination, with chicken-pox virus, by scarifying comb and wattles and rubbing in virus. Two of these developed slight scabs on the combs about the tenth day, while the others have developed no signs of infection.

A nearby farm flock, consisting of 50 birds, had an outbreak of chicken-pox, and it was decided to vaccinate these birds to see what effect the pigeon-pox virus would have upon birds already sick. The owner volunteered the use of his flock for this purpose, as the flock was producing only 4 to 6 eggs per day. All of these 50 hens were vaccinated on January 14, including 8 with lesions on the combs. On January 24, or ten days following, the 8 birds with chicken-pox lesions were all well and egg-production was increasing daily. The owner informed us, on February 12, that no new cases developed and the entire flock was well and had reached 50 per cent production.

Conclusions

Pigeon-pox virus vaccine, prepared as described, is a very satisfactory immunizing agent for preventing natural infection of chicken-pox.

This product was not 100 per cent efficient in immunizing against artificial infection.

Pigeon-pox virus vaccine seems to have no ill effects upon the birds, nor was any decrease noted in egg-production following its use.

References

[1]Johnson, W. T.: The effect of fowl- and pigeon-pox virus vaccination on egg-production. Jour. A.V.M.A., lxxviii (1931), n. s. 31 (1), pp. 98-101.

[2]Zwick, W., Seifried, O., and Schaaf, J.: Tierarzt. Wchnschr., xliv (1928), pp. 433-435. Cited Exp. Sta. Rec., lx (1929), 7, p. 671.

[3]Doyle, T. M.: Immunization of fowls against fowl pox by means of pigeon-pox virus. Jour. Comp. Path. & Therap., xliii (1930), 1, pp. 40-55.

EXPERIMENTS TO DETERMINE THE EFFECT OF SODIUM HYDROXID AND CALCIUM HYDROXID ON THE VIRUS OF HOG CHOLERA

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In view of the fact that sodium hydroxid has proved to be a most efficient disinfectant against the virus of foot-and-mouth disease, it was thought that it might also prove to be effective for the virus of hog cholera and thus furnish a cheap and effective agent for the disinfection of stockyards, pens, or other infected premises.

The following experiments were designed with the view of determining the concentrations of sodium hydroxid and milk of lime required to kill the virus of hog cholera in virus blood within a comparatively short interval of time. The combination of sodium hydroxid and milk of lime was used, as it is desirable, in the carrying out of official disinfection, to have some means of determining the thoroughness of the work and this would be indicated by the lime, which would have the effect of a whitewash. Furthermore, the lime also tends to preserve the sodium hydroxid, preventing its conversion into carbonate. It was found by experiment that 2 per cent milk of lime showed up quite well on sprayed surfaces and in most of the experiments milk of lime was used in this concentration in combination with varying concentrations of sodium hydroxid.

In carrying out the tests, a 10 per cent solution of sodium hydroxid and a 20 per cent suspension of milk of lime were used. The disinfectants were added to defibrinated virus blood in small glass dishes and rapidly incorporated with the blood by vigorous stirring. The mixtures of blood and disinfectants were then allowed to stand at room temperature for varying intervals of time, at the end of which susceptible pigs were injected with the mixtures in order to determine whether the virus had been killed by contact with the disinfectants. Each pig was injected with 5 cc of the mixtures of virus blood and disinfectants. The virulence of the different lots of virus blood was checked in each experiment by the injection of control pigs with virus blood to which no disinfectant was added.

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It was found that when a 10 per cent solution of sodium hydroxid was added to virus blood so as to give a concentration of 1 per cent NaOH, the blood was quickly changed to an almost clear, thick, syrupy liquid. When sufficient milk of lime was added to virus blood so that the blood contained 2 per cent milk of lime, the blood underwent marked thickening and was completely gelatinized at the end of two hours. When the sodium hydroxid solution and milk of lime were both added to virus blood so that the blood contained 1 per cent NaOH and 2 per cent milk of lime, the blood thickened perceptibly but not so much as when sodium hydroxid alone was added in a concentration of 1 per cent.

The results of the tests are shown in table I.

In addition to the foregoing experiments, another experiment was carried out, in which dry calcium hydroxid was used in place of milk of lime. In this experiment, 5 cc of a 30 per cent solution of sodium hydroxid and 1 gram of dry calcium hydroxid were added to 45 cc of virus blood. The calcium hydroxid was first thoroughly mixed with the sodium hydroxid and the mixture of the two was added to the virus blood. This gave a mixture which contained approximately 3 per cent NaOH and 2 per cent $Ca(OH)_2$. Each of two pigs, weighing 45 and 60 pounds respectively, was injected with 5 cc of this mixture, one pig being injected after the mixture had stood at room temperature for 15 minutes and the other pig after an interval of 30 minutes had elapsed. These pigs remained well following the injections and proved to be susceptible to hog cholera upon subsequent exposure. This experiment serves to confirm the results obtained in experiment 5 in the table, where the virus was killed after contact for 15 minutes with 3 per cent NaOH and 2 per cent milk of lime.

While there are slight discrepancies in some of the tests shown in the table, due probably to variations in the susceptibility of the pigs, it will be noted that pigs receiving virus blood containing 2 per cent sodium hydroxid and 2 per cent milk of lime contracted hog cholera, indicating that the virus had not been killed, whereas pigs injected with virus blood containing 3 per cent sodium hydroxid in combination with 2 per cent milk of lime remained well. It would seem, therefore, that these tests show quite clearly that 3 per cent sodium hydroxid in combination with 2 per cent milk of lime is effective in destroying the virus of hog cholera in virulent blood within a comparatively short time, that is, within fifteen minutes.

TABLE I—*Experiments to determine the effect of sodium hydroxid and calcium hydroxid*

TABLE I—*Experiments to determine the effect of sodium hydroxid and calcium hydroxid on the virus of hog cholera.**

EXPERIMENT	PIG	WT. (LBS.)	DATE INJECTED	MATERIAL INJECTED (EACH PIG WAS INJECTED WITH 5 CC OF MIXTURE)	% NaOH IN MIXTURE	% MILK OF LIME IN MIXTURE	VIRUS IN CONTACT WITH DISINFECTANTS	RESULTS OF INJECTION
1	254	50	6–26–29	18 cc virus blood + 2 cc NaOH solution	1	..	2 hrs.	Remained well
	256†	50		18 cc virus blood + 2 cc NaOH solution	1	..	19 hrs.	Remained well
	255	50		16 cc virus blood + 2 cc NaOH sol. + 2 cc milk of lime	1	2	2 hrs.	Remained well
	257	60		16 cc virus blood + 2 cc NaOH sol. + 2 cc milk of lime	1	2	19 hrs.	Remained well
2	35	85	8–2–29	18 cc virus blood + 2 cc NaOH solution	1	..	30 min.	Remained well
	38	90		18 cc virus blood + 2 cc NaOH solution	1	..	1 hr.	Developed h.c.
	37	90		18 cc virus blood + 1 cc NaOH sol. + 1 cc milk of lime	0.5	1	30 min.	Developed h.c.
	40†	80		18 cc virus blood + 1 cc NaOH sol. + 1 cc milk of lime	0.5	1	1 hr.	Remained well
	36	85		16 cc virus blood + 2 cc NaOH sol. + 2 cc milk of lime	1	2	30 min.	Remained well
	39†	85		16 cc virus blood + 2 cc NaOH sol. + 2 cc milk of lime	1	2	1 hr.	Remained well
3	87	40	9–6–29	16 cc virus blood + 2 cc NaOH sol. + 2 cc milk of lime	1	2	5 min.	Developed h.c.
	88	50			1	2	5 min.	Developed h.c.
	89	55			1	2	10 min.	Developed h.c.
	90	65			1	2	10 min.	Developed h.c.
	91	45			1	2	15 min.	Developed h.c.
	92	65			1	2	20 min.	Developed h.c.
	93	65			1	2	30 min.	Developed h.c.
4	112	85	9–17–29	14 cc virus blood + 4 cc NaOH sol. + 2 cc milk of lime	2	2	5 min.	Developed h.c.
	113	85			2	2	10 min.	Developed h.c.
	114	85			2	2	15 min.	Developed h.c.
	115	85			2	2	30 min.	Developed h.c.
5	149†	45	11–1–29	12 cc virus blood + 6 cc NaOH sol. + 2 cc milk of lime	3	2	15 min.	Remained well
	151†	50		12 cc virus blood + 6 cc NaOH sol. + 2 cc milk of lime	3	2	30 min.	Remained well
	150†	50		10 cc virus blood + 8 cc NaOH sol. + 2 cc milk of lime	4	2	15 min.	Remained well

*In these experiments a 10% solution of NaOH was used and a 20% suspension of milk of lime.
†Proved to be susceptible to hog cholera upon subsequent exposure.

Note: Five of the pigs which developed hog cholera were killed for virus, three were killed when moribund or worthless, two died, and three recovered in poor condition.

A LARYNGOTRACHEITIS SYNDROME IN WILD GOOSE ASSOCIATED WITH PNEUMOMYCOSIS*

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Acute laryngotracheitis and clinically analogous diseases have been responsible for increasing losses during the past decade in farm flocks as well as young chicks from hatcheries. A similar clinical and probably the same disease has menaced the shipping of middle western poultry to eastern market centers. Investigations have been conducted in many different outbreaks on the etiology and epizoology of the malady, looking to the prevention of this disease. Measures of prevention and control are to be largely gained through sanitary management.

The studies also have thrown some light on the nature of the syndrome referred to as laryngotracheitis. A clinical diagnosis of laryngotracheitis does not imply the causative factor or factors in each outbreak. An invasive filtrable virus-like agent, though not consistently demonstrated, as well as pathogenic bacteria in the upper respiratory tract of naturally infected fowls, have not satisfactorily explained all clinical outbreaks of the disease. Clinical symptoms in the subacute malady may not be easily recognized, while the gross lesions in the acute malady are variable and not always in keeping with the symptoms displayed. It has, therefore, been repeatedly suggested that the acute syndrome is not a specific entity and may be associated with more than one cause.

A chicken-pox laryngotracheitis (encountered only occasionally) may be differentiated from the prevailing type in a majority of cases by the character of the gross lesions. However, pathognomonic inflammatory lesions, traceable to other than chicken-

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pox virus infection, in the upper respiratory tract, are not so easily recognized. Clinical laryngotracheitis infection in chickens (exclusive of chicken-pox) of different ages (1 month to 2½ years) as well as pea fowls and pigeons has been recognized. The purpose of this report is to describe the occurrence of a spontaneous laryngotracheitis syndrome in a wild goose associated with a pulmonary mycosis. The goose was submitted to the laboratory for diagnosis on March 30, 1931.

FIG. 1. Wild goose showing acute laryngotracheitis syndrome. Respiration became labored and audible mucous râles increased as the disease progressed. The disease was diagnosed on the basis of clinical symptoms as acute laryngotracheitis.

The only clinical symptom noticed on arrival was an occasional mucous râle in the larynx, suggestive of the primary stages of an acute respiratory syndrome. The affected goose was promptly segregated from the remaining healthy fowls on the farm, and according to the owner was the only fowl affected among a large group of domesticated and wild water fowls at the time the sick goose was delivered for diagnosis. The suspicious respiratory symptoms prompted holding the goose for observation. Over a

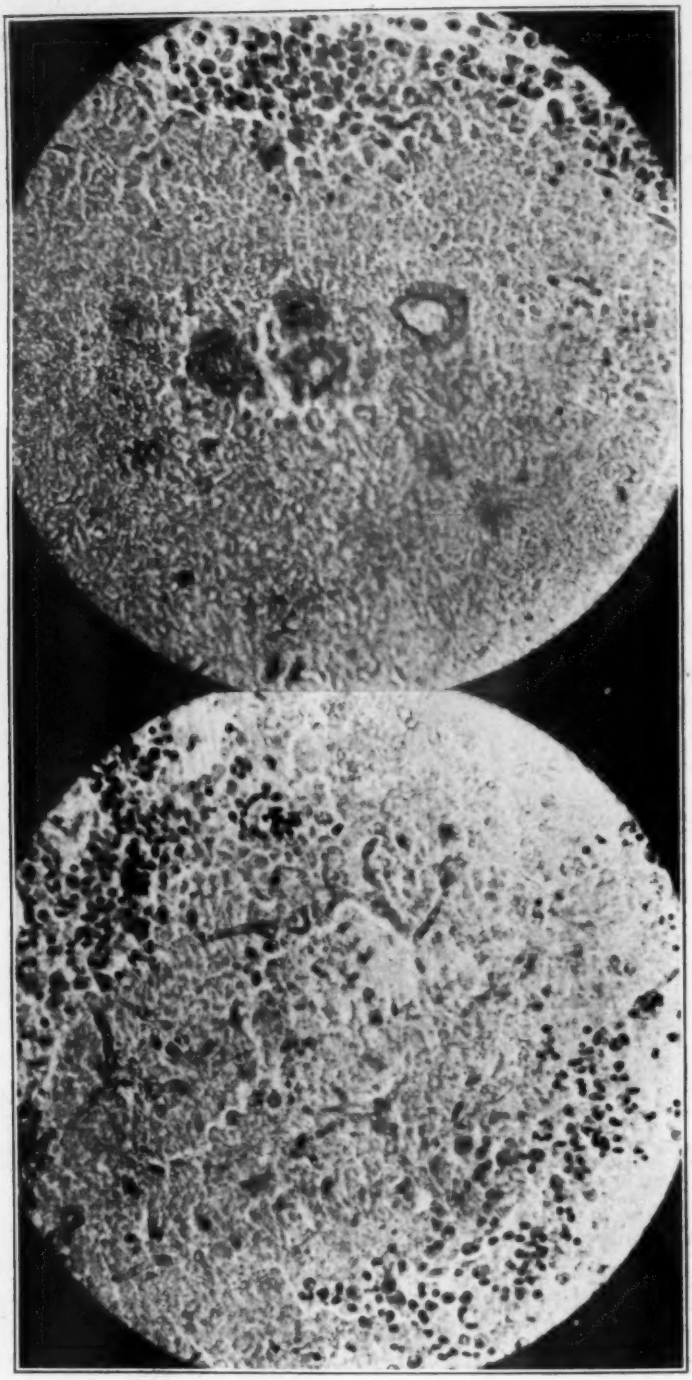

FIG. 2. (Above). Four mycotic gonidiospores in the bronchial exudate. Polymorphonuclear leucocytes and lymphocytes in upper part of the field.

FIG. 3. (Below). Branching segmented mycelia in the bronchial exudate. Lymphocytes, erythrocytes, and a few polymorphonuclear leucocytes surrounding the mycelia.

four-day period the respirations became labored, and open-mouth breathing was common (figure 1). Audible laryngeal and bronchial râles were pronounced as the disease advanced. The clinical symptoms were quite analogous to acute laryngotracheitis in fowls.

Autopsy

The affected goose at autopsy showed a few scattered petechial hemorrhages in the mucous membrane of the larynx. A rusty brown-tinged mucus, slightly adherent to the mucous membrane, extended from the larynx down the trachea to the bronchi. In the bronchi small particles of yellowish necrotic exudate were found. The lungs were mildly congested. The heart, liver and kidneys were normal. The duodenum showed a catarrhal enteritis accompanied by a heavy infestation of tapeworms.

A histopathological study of the lung revealed foci of mycotic pneumonia (figure 2). Lesions in the bronchioles consisted of masses of branching segmented filaments of mycelia in the exudate containing erthrocytes, lymphocytes and polymorphonuclear leucocytes. In advanced lesions the epithelial cells lining the bronchioles were displaced by fibrous connective tissue. In other less inflamed bronchioles the epithelium was intact, though the lumen was occluded by an exudate consisting of fibrin and polymorphonuclear leucocytes. The radiating mycotic filaments penetrated the exudate (figure 3).

Transmission Experiments

Direct inoculation experiments were carried out on five chicks (1 month of age) using a saline suspension of the unaltered exudate from the larynx and trachea. The suspension was administered to 2 chicks intravenously in 1- and 2-cc doses each; 2 chicks received intralaryngeally .03 and .06 cc each, respectively, while the fifth was given 2 cc intraperitoneally. The inoculated chicks remained healthy.

The laryngeal and tracheal exudate was emulsified in sterile saline and passed through a Berkefeld N filter. The bacteriologically sterile filtrate was administered intravenously in 2-cc amounts to four healthy chicks (1 month of age). No symptoms or lesions of laryngotracheitis developed in the chicks.

Summary

A laryngotracheitis syndrome in a wild goose, associated with gross exudative lesions of the respiratory tract and mild con-

gestion of the lungs, proved to be a mycotic pneumonia. The mycosis was not communicated by direct swab of the larynx and trachea or by sterile filtrates prepared from the exudate in the respiratory tract when administered to healthy chicks one month of age.

STREPTOCOCCUS PYOGENES MENINGEAL ABSCESS CAUSING PARAPLEGIA

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Subject: Poland China sow pig, five weeks old, in good condition, as judged by weight and general appearance.

History: Pig was apparently normal until the evening of March 26, 1931, when the herdsman observed lameness or soreness in the hind quarters, as exhibited by unsteady gait, shortening or in some instances complete loss of the second half of the

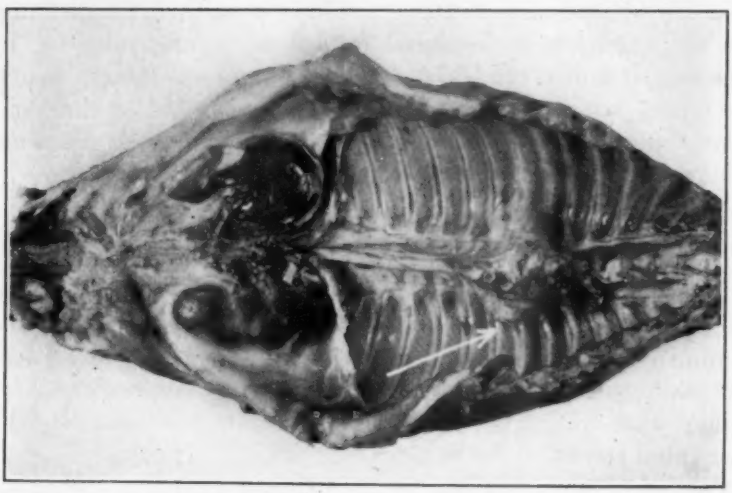

Fig. 1. Vertebral abscess (shown by arrow) in young pig, caused by a hemolytic streptococcus.

phase of flight, and incoördination of movements causing the animal to veer unsteadily from side to side while walking.

Symptoms: This pig was examined on March 27, 1931. Complete bilateral paraplegia of the posterior limbs was observed. The syndrome was clinically analogous to the prevailing posterior

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paralysis of mature hogs due to deficiency of calcium or phosphorus in the diet. Sensation was absent in both limbs and over the regions of the thigh and croup.

Gross pathology: At autopsy all body organs were apparently normal. An enlargement emanating from the ventral surface of the sixth, seventh, eighth and ninth thoracic vertebrae and the proximal ends of the corresponding ribs was noted (figure 1). The pleura and connective tissue were distended in this area, forming a pouch or cavity which, upon incision, discharged a thick, yellow pus. On exploration the abscess was found to extend to the meninges, exerting pressure on the spinal cord. The pyemic lesion had apparently been of long standing, with symptoms of paraplegia appearing only as pressure on the spinal cord developed.

Bacteriological findings: Direct smears of the abscess revealed a great number of cocci and diplococci. Cultures of the pus on blood-agar plates revealed a hemolytic streptococcus with sugar fermentation similar to *Streptococcus pyogenes.*

Diagnosis: Streptococcus pyogenes suis abscess, causing pressure on the spinal cord in the thoracic region.

INTESTINAL STENOSIS IN A SHEEP, POSSIBLY FOLLOWING INTUSSUSCEPTION, WITH AUTOSURGERY

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Department of Animal Pathology, Michigan State College

The animal is a three-year-old ewe, brought in for autopsy. The owner presented the following history. About one month before death, she suddenly became very sick. After three or four days, she appeared practically normal again, but about two weeks later she began to lose appetite, gradually became weaker and died.

At autopsy the only changes of note are found in the abdomen. The visceral peritoneum shows considerable congestion, especially the mesentery. There is marked peritoneal thickening over much of the small intestine, suggesting previous peritonitis. The first three compartments of the stomach are negative. The abomasum is somewhat distended with liquid ingesta. In the first 25 feet of the small intestine, distention with fluid ingesta becomes increasingly greater. At the largest point the diameter is 4 cm. This distention ends suddenly in a marked constriction, caused

by a thick connective tissue scar which causes almost complete obstruction of the lumen of the intestine. From this point the remainder of the intestine is practically empty and shows no gross changes. The liver and kidneys show some degenerative changes.

FIG. 1. Note the extreme dilatation of the intestine at A, the thick, contracted cicatrix at B, and the normal intestine at C.

The history and autopsy findings indicate that an intussusception was followed by autosurgery and apparent recovery of the animal. Subsequent contraction of the connective tissue scar then gradually led to stenosis of the intestine and death of the animal.

DIABETIC COMA OF FEED-LOT SHEEP

Preliminary Report

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J. J. REID, *Laboratory Director,*
Live Stock Sanitary Commission, and

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Fort Worth, Texas

Recent investigation has demonstrated the occurrence of diabetic coma in sheep, in feed-lots where the rapid method of fattening is practiced. As no reference to this condition among sheep has been found in the literature, we have no accurate figures available with which to establish a definite death loss, but it is believed that a 3 per cent average would be a very conservative estimate.

Death loss from this condition has been observed for the past six or seven years. This loss was exceedingly heavy at times, and various authorities were called to investigate and to offer suggestions for relief.

Death occurred with such rapidity in most of these cases, that postmortem examination was the most available method of study. The causes of death were attributed by many investigators to various disease conditions. Among these might be mentioned hemorrhagic septicemia, pulmonary edema, indigestion, mineral deficiency, and forage poisoning.

It has been observed that the death loss may vary considerably from time to time, depending upon certain predisposing factors, such as cold, damp or rainy weather, also the amount of carbohydrates furnished in the ration.

The symptoms are varied in the affected sheep, but without exception there is a very sudden onset and a rapid termination, death usually occurring within from one to three hours. Apparently healthy, fat sheep may start suddenly, spring as high as possible into the air, falling on their side in occasional convulsions with the head thrown back, the lips twitching and champing of the jaws producing a frothy saliva. Sometimes the affected sheep will exhibit symptoms of botulism, pushing against the fence with their heads, apparently unmindful of surrounding

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conditions. They may stand with the head excessively elevated and work their lips convulsively. The breathing is labored and intermittent, giving the observer the impression that they are unable to acquire sufficient air. They soon lose their equilibrium, staggering, falling and showing occasional convulsions. A state of coma rapidly ensues and death follows. The temperature of the affected animal varies. Those undergoing violent convulsive movements show elevation of temperature, though not so high as is found in acute febrile diseases.

Hundreds of sheep dying in comatose condition have been autopsied, but no consistent findings have been presented to explain the death of the animals. Failure to find any pathological lesions while conducting autopsies necessitated further investigation. It might be mentioned that previous investigators had tried unsuccessfully to demonstrate that the conditions were due to microörganisms.

With these facts in mind, February 14, 1931, we obtained blood and urine for examination from a sheep that was in coma. The urine sugar was 0.98 per cent; the blood sugar was 250 milligrams per 100 cc of blood; also there was a distinct trace of acetone and diacetic acid in the urine.

February 16, we examined blood and urine from a normal stocker sheep, a normal fat sheep and a fat sheep in coma, with the following results:

Stocker sheep	Urine sugar—Negative Blood sugar—66.6 mgm/100 cc
Normal fat sheep	Urine sugar—Negative Blood sugar—79.8 mgm/100 cc
Fat sheep in coma	Urine sugar—3.48 per cent Blood sguar—300 mgm/100 cc

Several of these animals were treated with insulin. After blood and urine specimens were secured, 40 units of U 40 insulin was injected intravenously, followed by 200 cc of normal saline solution. Thirty minutes later this dose of insulin was repeated. Two hours following the first insulin injection, blood and urine specimens were again collected and examined. It was found that the blood and urine collected before treatment were abnormally high in sugar; while specimens collected after treatment showed the blood sugar content of the animal greatly reduced with the urine entirely negative for sugar.

One of the animals so treated made a complete recovery after remaining in a semi-comatose condition for several days, whereas other animals, treated as above mentioned, passed from the state

of diabetic coma into insulin coma. Still other animals were given both insulin and glucose, a treatment frequently used in human diabetic coma. These animals died.

Due to the rapid course of this disease in sheep, we are of the opinion that treatment with insulin is not practical because of the necessity of frequent examinations of the blood to prevent the transition from diabetic coma to insulin coma.

Thus far, the only practical preventive measure is a reduction in the amount of carbohydrates supplied in the ration within safe limits. The feeding period is thereby increased to approximately one-fourth longer than that of the rapid method. The death loss is negligible in such a procedure.

PERITONEAL TUMORS IN THE FOWL

By M. W. Emmel, *Auburn, Ala.*

Department of Animal Pathology, Alabama Polytechnic Institute

Tumors of the peritoneum in the fowl, while not of common occurrence, have been found in three birds during the past year. All of these birds were killed for autopsy, two of them having a ruptured egg-yolk in the peritoneal cavity, while the third had a heavy infestation of *Davainea proglotina*. In the former two birds the ruptured egg-yolk was probably the exciting factor. The intestinal wall was firm, thickened, and whitish gray in color. The serosa was studded with numerous nodules ranging in size from that of a small pin-head to enlargements 4 mm. in diameter. The smaller nodules were visibly flattened, with some tendency towards becoming diffuse. Upon gross examination these tumors seemed to involve chiefly the serosa. In each case the mesentery was whitish gray, firm, and irregularly thickened, on many occasions reaching a thickness of 2.5 mm. although there was no appearance of the tissue being nodular.

Only the duodenum of the third bird was affected. The intestinal wall was considerably thickened and presented the appearance of coarse, regular, flattened nodulations, with occasional large nodules. At the time, however, it did not occur that the thickening of the intestinal wall might be due to a neoplasm involving the serosa. As thickening of the intestinal wall is of rather common occurrence in birds affected with enteritis, the thought suggests itself that neoplasms involving the mesothelium may be of more common occurrence that would be otherwise

expected. The exciting factor in this latter case is doubtful, although the evidence suggests that the tumors may have had their origin in the subserous lymphatic vessels and may have been caused by the absorption of toxic substances through these channels.

A microscopic study was made of the neoplasms in these three birds. Stengel and Fox[1] describe a tumor of the peritoneum, and again Karsner[2] describes tumors of the peritoneum and pleura in the human which resemble the general microscopic structure of these tumors in the fowl.

The serosa of the duodenum of the bird infested with *Davainea proglotina* was generally thickened to the extent of 1 mm. The

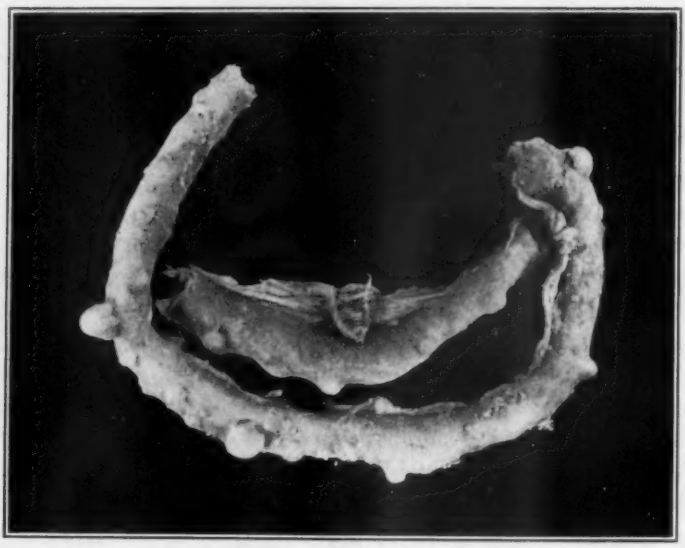

FIG. 1. Tumors of the peritoneum in the fowl.

outer surface was smooth but very sharp undulations occurred which macroscopically gave the appearance of coarse nodulations. The epithelial cells of the serosa were arranged in loose axial strands. However, the cells were slightly more closely packed at the base, where there seemed to be some overlapping with a reaction of the subserous connective tissue, distinctly more packed in the central zone and only slightly so at the surface. The epithelial cells as a general rule were large, with clearly defined nuclei and sharply differentiated nucleoli. There was a slight general edematous condition throughout, which as a rule

was more marked in areas where the strands of cells were most loosely arranged.

The subserous connective tissue was slightly hyperplastic and in many areas showed slight extension into the new growth of tissue in the serosa. Perivascular foci of hyperplasia were of occasional occurrence in conjunction with the intermuscular lymph-channels. Large flattened nodules were occasionally observed in which the arrangement of epithelial cells was no longer in strands but occurred more in foci, between which active proliferation of connective tissue occurred and which at times tended to become diffuse. These larger nodules were covered with a superficial layer of epithelial cells, loosely arranged, in concentric strands with slight edema in evidence. There was some involvement of the longitudinal muscle coat.

Most of the epithelial cells, both in the foci and those occurring singly, in the mass of the nodule, were swollen often to three or four times their normal size and in a state of degeneration, principally cloudy swelling. The nuclei and nucleoli of these cells stood out very distinctly. A few of these nuclei showed definite pyknosis. Numerous large intercellular acini and accumulations of eosinophiles occurred throughout these larger nodules. In general the longitudinal muscle coat was in a state of cloudy swelling with a few scattered foci of fatty degeneration, and this was true also in those areas in which the muscle tended to become an integral part of the larger nodulations.

The general structure of the new growths in the serosa of the birds in which ruptured egg-yolks were found in the peritoneal cavity was very much the same as that described above. However, while the smaller nodules were distinctly flattened, the larger ones were more definitely nodular, often reaching a diameter of 4 mm. There was also very much less reaction in the serosa between the nodules and less tendency to axial strands of epithelial cells. The larger nodules showed much more proliferation of connective tissue which often occurred in perivascular whorls. In these larger nodules the proliferation of connective tissue often extended into the mucosa, even involving the villi at times. In such instances there was much mucoid degeneration of the glandular as well as the surface epithelium. The mesentery of these two birds was irregularly thickened, often as much as 2.5 mm. The general reaction here was similar to that found in the intestinal wall, the increased thickness being caused by proliferation of connective tissue. Central foci of necrosis were more

numerous, as was also the occurrence of irregular accumulations of eosinophiles.

From our study it appears that the origin of such tumors in these latter instances was in the mesothelium, and that the egg-yolk was no doubt the exciting factor. In the other bird it appears that the origin may have been in the subepithelial lymph-channels. Whether such tumors arise from the mesothelium or endothelium, it appears that both may become finally involved in the process.

REFERENCES

[1]Stengel, A., and Fox, H.: A Textbook of Pathology (W. B. Saunders Co., Philadelphia and London, 1927), p. 765.

[2]Karsner, H. T.: Human Pathology (J. B. Lippincott Co., Philadelphia and London, 1929). pp. 589 and 657.

POISONING IN CHICKENS WITH WHORLED MILKWEED*

By H. W. CAMPBELL, *Petaluma, Calif.*

Division of Animal Industry, California Department of Agriculture

The narrow-leafed whorled milkweed, *Asclepias Mexicana*, which is widely distributed in California and other western states, is known to be poisonous to sheep, cattle and horses. A review of available literature failed to reveal reference to cases of poisoning with this plant in poultry. A very good description of the plant, with symptoms of poisoning in large animals, may be found in "Stock Poisoning Plants of the Range," U. S. D. A. Bulletin 1245.

A condition developed in a flock of pullets near Santa Rosa, California, in which the symptoms were not characteristic of any of the diseases generally recognized in poultry. A number of birds from this flock were brought for diagnosis to the State Division of Animal Industry's Poultry Pathological Laboratory in Petaluma, California. The condition was diagnosed as a nervous affliction of unknown cause or causes.

The writer visited the ranch in an effort to determine the possible cause of the losses. It was observed that the condition was occurring in a flock of 700 White Leghorn pullets, eight weeks old. Cockerels of the same brood and other birds on the ranch were not affected. The management of the pullets and cockerels was identical excepting that the pullets had the run of a small

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yard, while the cockerels were confined to the house. Adult birds on the ranch were allowed the run of a small orchard.

Only a few birds were affected at a time. Approximately thirty of the pullets had shown symptoms in the three weeks prior to the visit. About 50 per cent of these had died while the remainder had recovered after manifesting symptoms over a period ranging from two days to one week. The first indication was lameness, which rapidly developed into a complete loss of muscular control. The head was twisted and drawn back. The eyes were bright and the comb did not lose color at any time.

Upon investigation it was found that a quantity of whorled milkweed was growing in the pullet yard. From the character of the plants, it was evident that the birds had been eating it to a considerable extent. The weed was present also in the orchard, but the adult birds seemed to leave it alone.

A quantity of the weed was gathered and brought to the laboratory to determine whether or not it was toxic to poultry. Four stalks were chopped up and placed before two four-month-old pullets at 4:00 p. m. They ate it readily. At 8:00 a. m. the following day, both birds were completely collapsed. The symptoms in these birds were identical with those found in the field cases. The condition was not a true paralysis, but seemed to be an over-stimulation of the motor nerve centers with a complete loss of coördination. The birds lay on their sternums or sat on their hocks. The head was drawn back with neck bowed to the front in the manner of a pouter pigeon. At times the head would be alternately extended and retracted at frequent intervals. At other times the head would be rotated until it rested on the back of the bird directed toward the tail. When suddenly disturbed, the birds would fall over backward and struggle violently with convulsive movements of the legs and wings. The symptoms of the disease were quite violent for three days, and then gradually became less severe, although there were occasional relapses into the original manifestations. After two weeks, both birds had apparently recovered.

An adult White Leghorn hen was then fed 20 grams of fresh ground roots of the plant. Another hen was fed the same amount of fresh ground leaves, stems and blossoms. Both birds showed violent symptoms characteristic of the condition. The bird which had consumed the top parts of the plant died in a coma on the fourth day, while the other gradually recovered and was

normal after ten days. No treatment was attempted. Autopsy was negative.

The adult birds on the ranch having access to a large range did not eat enough of the weed under normal conditions to cause poisoning, although there was a considerable quantity growing in the orchard in which they ranged. However, when the owner was advised of the cause of the trouble in the pullets, he decided to eradicate the plant completely from his premises. While in the process of digging it out, the hens were attracted to the newly broken ground and consumed a sufficient quantity of roots and tops to cause a severe loss. In this connection it is of interest to note that the plant is very difficult to eradicate. It is deeply rooted and, when the roots are broken, a new plant sprouts from each root portion wherever it is covered with soil.

It is not possible to estimate how extensive losses are from this cause. The symptoms are such that it may easily be confused with paralysis, botulism, or other toxemic conditions.

COW ADOPTS COLT

By D. E. Bleecker, *Columbus, Wis.*

The accompanying photograph depicts an unusual family arrangement. The colt's mother, owned by one of our clients, died when the youngster was but a few days old. With very

Fig. 1. Unusual family arrangement.

little difficulty on our part, the cow was persuaded to adopt the orphan. This interesting phenomenon prompted the sending of the photograph for reproduction in the Journal.

THE BOBWHITE QUAIL—ITS HABITS, PRESERVATION AND INCREASE. By Herbert L. Stoddard. 559 pages, with 64 pages of half-tone illustrations, 4 color plates, 32 text figures and 51 tables. Charles Scribner's Sons, New York, 1931. Regular edition, $6.00; limited edition, $60.00.

At the present time there is a greatly increased interest in game birds in general and in the bobwhite quail in particular. Game refuges and game farms are increasing or expanding and programs of restocking are being carried out. Even more significant is the fact that game birds are coming to be regarded as a secondary farm crop, with one state (Wisconsin) taking the lead in paying the farmer for the crop. In view of this increased interest and these new activities, veterinarians will find more and more that such birds as the bobwhite quail will come within the sphere of their professional pursuits; the present book will accordingly hold much interest and significance for them.

The book deals with a popular subject—the habits, preservation and increase of a bird which ranks high in the esteem of bird lovers and of sportsmen; on the whole, the subject matter is handled in a popular style, but with this distinction—that it sticks to facts—facts obtained through observations made by critical investigators. The study was financed by a group of public-spirited sportsmen and was conducted by the Bureau of Biological Survey of the Department of Agriculture, with the coöperation of other specialists who are eminent in their respective fields. The report embraces a great fund of material presented in an enlightening and stimulating form for the reader whose interest in the subject is general; however, with sufficient attention to detail to assure its usefulness for those concerned with a special phase of the subject. The study is without doubt more comprehensive in its scope and more highly unified than any previous study made of any bird.

Chapters which are most noteworthy for the veterinarian include Food and Feeding Habits, Relation of Agricultural

Activities to the Bobwhite, Quail Preserve Development and Management, Control of Natural Enemies, Artificial Propagation of the Bobwhite, and of even greater significance to veterinary medicine, Mortality in the Bobwhite, Internal Parasites and Parasitic Diseases, External Parasites, and Non-Parasitic Diseases of the Bobwhite.

The problem of the relation of live stock to quail and other upland game birds is a subject of vital importance at present. There have been mutual benefits in the relationship of quail and of agriculture as the latter developed along simple, or what are now considered primitive lines. Quail have profited by an increased food supply; agriculture has profited by the aid of the birds in combating insect and weed pests However, civilization has upset the adjustment of quail to nature by bringing in cats and dogs and, perhaps even more important, poultry with its diseases, and more recently by the development of the modern highly intensive and mechanized type of agriculture. As regards live stock, cattle, mules, horses, sheep and goats destroy nests and reduce the food supply of the birds, especially as regards legumes, by trampling or grazing. Hogs, on the other hand, if properly handled, are an advantage to the quail population; by their rooting they make such food as tubers available to the birds and their open-mouthed chewing of corn, acorns and nuts leaves bits for the quail which follow in their wake. Moreover, vegetation which is favorable to quail grows up where hogs have rooted.

The chapter on internal parasites and parasitic diseases was prepared by Eloise B. Cram, Myrna F. Jones and Ena A. Allen, of the U. S. Bureau of Animal Industry. Four protozoan diseases are recorded from the bobwhite, namely, coccidiosis, blackhead, trichomoniasis and malaria. Coccidiosis and tapeworm infestations proved to be the two greatest stumbling blocks in Stoddard's artificial rearing of quail; the discussions of the modifications which he made in the methods of rearing quail to overcome these diseases should prove of great practical value to others who are raising domestic or game birds. Tapeworms are frequent in quail raised in captivity with chickens as foster parents and in quail found at large but in or near areas ranged by poultry. One species is apparently native to quail, occurring irrespective of their proximity to domestic fowls. Five species of tapeworms and 16 species of roundworms are reported. For both groups of parasites a general discussion of the nature of the

parasites, their life histories, control methods and treatment is followed by semi-technical descriptions of each species.

The chapter on external parasites, written by Stoddard, discusses the lice, mites, ticks and fleas found on quail, identifications of which were made by H. E. Ewing. Ticks were found to cause considerable annoyance and chiggers were noted at times to produce marked irritation, with resulting swollen areas.

The non-parasitic diseases include "foot disease," bird-pox, dry gangrene, chicken-pox, "nutritional roup," aspergillosis, "quail disease" and tularemia. No serious disease outbreaks were noted by Stoddard among quail in the wild but among birds which were more or less closely associated with poultry, losses occurred from chicken-pox and other poultry diseases. "Quail disease," as yet unknown in the wild, is probably more frequently met with than any other disease in the artificial rearing and handling of quail; it is seen usually among birds which are confined in "stale" pens or in shipping-crates. Stoddard's success in controlling this disease will greatly encourage others who are raising game birds. Tularemia is discussed as a potential danger, on the basis of the reports of Parker and of Green and Wade, which show the susceptibility of quail to this disease; rabbit ticks, which are found very frequently on quail, furnish a possible means of transmission of the disease.

The index to the book is complete and satisfactory, making quick reference possible. The veterinarian who is interested in quail and other game birds will find this book a valuable reference.

E. B. C.

INVERTEBRATE ZOÖLOGY. Harley Jones Van Cleave. 2nd edition. 282 pages, with 126 figures. McGraw-Hill Book Co., New York, 1931. Price, $3.00.

The second edition of Van Cleave's Invertebrate Zoölogy is quite well gotten up and is written in a lucid manner. While not an exhaustive treatise, it gives the student a more or less comprehensive survey of the invertebrates. The present edition shows a decided improvement over the earlier edition. Much new material and many new illustrations have been added, and an outline of the classification of each phylum has been appended to each chapter; this greatly increases the usefulness of the book.

There are certain alterations which should be made in later editions. In his discussion of the liver fluke the author has incorporated old material which is still current in text-books but is

contrary to the findings of late years. Such statements as that the cercariae crawl upon grass or vegetation before encystment, that the cysts are calcareous, and that the young flukes in the intestine reach the liver through the opening of the bile duct are statements which are not in keeping with the latest information.

It would have been better to refer to the lateral lines of nematodes as lateral chords, and to have made some exceptions to his description of the excretory system of nematodes. Not all nematodes have the same type of excretory system. The statement that "locomotor organs are wholly lacking" in the nematodes fails to take cognizance of such exception as *Draconema*, and the statement that "distinctive free larval stages are lacking" is not clear but seems to imply something that is not true; certainly there are free larval stages which are distinctive and characteristic for various nematode groups.

It is not clear why *Mermis* should have been selected as one of the characteristic genera of free-living nematodes, since all of the Mermithidae are parasitic. It would have been just as proper to include *Necator* and other monoxenous nematodes having free-living larval stages in the same category. The scales in *Iota* are not scales, but are prolongations of the posterior margins of the cuticular annulations.

The above inaccuracies are more or less minor when one considers the scope of the work. By and large it is a volume which would be of use in the veterinary schools as a reference in furnishing background in zoölogy and in connection with the invertebrate hosts of worm parasites.

E. W. P.
B. G. C.

PUBLICATIONS RECEIVED

Studies in Dog Distemper. VI. Dog Distemper Antiserum. P. P. Laidlaw and G. W. Dunkin. Reprint from *Jour. Comp. Path. & Therap.*, xliv (1931), 1, pp. 1-25.

The Nature of Louping-ill. J. Russell Greig, A. Brownlee, D. R. Wilson and W. S. Gordon. Reprint from *Vet. Rec.*, xi (1931), 13, pp. 325-333.

Information for the Guidance of Field Men and Coöperators of the Bureau of Biological Survey Engaged in the Control of Injurious Rodents and Predatory Animals. Paul G. Redington and Stanley P. Young. (Misc. Pub. 115. U. S. Dept. Agr., Washington, D. C., April, 1931.) pp. 8.

Dog Distemer Antiserum. P. P. Laidlaw and G. W. Dunkin. Reprint from *Vet. Rec.*, xi (1931), 14, pp. 359-367.

If you are planing to attend the Kansas City Convention, make your reservations at the Hotel Baltimore without delay.

THE RELATION OF STREPTOCOCCI TO THE FILTRABLE VIRUS OF EPIZOÖTIC ENCEPHALITIS OF THE FOX. Edward C. Rosenow. Jour. Inf. Dis., xlviii (1931), 3, p. 304.

The consistent isolation of a streptococcus, with distinctive cultural, serologic and neurotropic properties from the brains of young foxes and dogs, less commonly from the brains of rabbits, which have succumbed to encephalitis following the inoculation of virus, is reported. From one strain of streptococcus isolated from the brain of a dog that succumbed to virus, and after 33 subcultures, a filtrable virus seemingly has been produced. Period of incubation, symptoms and lesions in the animals given artificially produced virus were similar to those given natural virus. The cultural characteristics, the serologic reactions, and the virulence of the streptococci from the animals given injections of the natural virus were similar to the reactions in the animals into which artificially produced streptococcal virus was injected. The streptococcus from the brains of animals that had been given injections of natural virus protected other animals against the artificially produced virus, and vice versa. The artificially produced virus resisted glycerination for periods ranging from 15 days to 6 months.

BACTERIAL FLORA OF THE LOWER RESPIRATORY SYSTEM OF NORMAL DOGS. H. Livingstone and W. E. Adams. Jour. Inf. Dis., xlviii (1931), 3, p. 282.

One-third of the dogs that appeared normal on external examination presented gross evidence of bacterial contamination of the tracheo-bronchial tree at autopsy. The results seem to indicate that there is constant presence of microörganisms in the peripheral lung tissue of normal dogs, with less frequent occurrence in the air-passages. Bacteria were present in the blood of four of eleven normal dogs. Contamination of the blood-stream was found more frequently in dogs showing gross contamination

of the tracheo-bronchial tree. The authors make no effort to explain the avenue of entrance of such organisms into the lungs and blood-stream other than to say that they were not aspired at death.

Anterior Pituitary Hormones. E. P. Bughee, A. E. Simond and H. M. Grimes. Endocrinology, xv (1931), 1, p. 41.

There are eleven distinct physiological functions claimed by various research workers for the anterior pituitary: (1) stimulation of growth, (2) stimulation of sexual development and ripening of follicles resulting in ovulation, (3) stimulation of lutein cell development resulting in prevention of ovulation by imprisoning the ova, (4) stimulation of sexual development by a substance which can be given by mouth, (5) stimulation of metabolism by increasing the specific dynamic action of food substances, (6) stimulation of the thyroid gland, (7) lowering of gaseous metabolism, (8) stimulation of water intake and output, (9) stimulation of lactation, (10) lowering of non-protein nitrogen in the blood, and (11) initiation of the bleeding of menstruation. Of these activities four may be due to a placental hormone rather than one elaborated by the anterior pituitary. Ten of these activities have been demonstrated by means of transplants of anterior pituitaries or by alkaline extracts. The hormones responsible are similar in their general chemical properties, such as stability to acid, alkali and heat. A growth hormone is clearly separate from one causing luteinization in ovaries, for the former is destroyed by 0.4 per cent tricresol, while the latter is not destroyed by the same treatment.

Tissue Specificity in Anthrax Infections. Victor Burke and Laverne A. Barnes. Jour. Immunol., xx (1931), 2, p. 173.

The ability of *Bacillus anthracis* to start an infection in various tissues of the body has been questioned. According to Besredka this organism can start an infection only in the skin. The experiments indicate that when *B. anthracis* is deposited subcutaneously and the needle puncture through the skin disinfected so that infection cannot start in the damaged tissue, the animal will die of typical anthrax infection, and secondly, that the characteristic subcutaneous gelatinous infiltration occurs at the point of deposit of the organisms and not at the point of puncture through the skin. The authors conclude that anthrax can start as an infection in the subcutaneous tissue and corroborate the

work of Blanc and Cammopteras, who demonstrated anthrax infection originating in the brain, and Lumiere and Montoloy, who produced infection in the peritoneal cavity.

THE SO-CALLED SPLENIC LESIONS IN CANINE RABIES. Rigney D'Aunoy and J. L. Beven. Jour. Inf. Dis., xlviii (1931), 3, p. 335.

Gross and microscopic study of the spleens of 100 rabid and 50 non-rabid dogs failed to establish any marked relative splenomegaly or microscopic splenic changes that could be considered pathognomonic for canine rabies.

SOME EFFECTS OF TEMPERATURE UPON DEVELOPMENT OF THE OOCYSTS OF COCCIDIA. E. R. Becker and H. B. Crouch. Proc. Soc. Exp. Biol. and Med., xxviii (1931), 5, p. 529.

The experiments show that non-sporulated oöcysts of *Eimeria magna* and *E. perforans* are killed in a water medium at a temperature of 51° C. and above for ten minutes. The optimum temperature for the development of the oöcysts in a 2 per cent potassium dichromate solution is 33° C.

PURIFIED (PROTEIN-FREE) VIRUS OF CHICKEN TUMOR NO. 1. Margaret Reed Lewis and William Mendelsohn. Amer. Jour. Hyg., xiii (1931), 2, p. 639.

Removing the protein from the tumor extract did not inactivate the tumor-producing agent. Twenty-two inoculations were made with extracts of nine tumors rendered protein-free by means of animal charcoal. Nineteen of these inoculations resulted in tumors at the site of injection. Ten inoculations were made with extracts of six tumors from which the protein had been removed by means of kaolin. All of them resulted in tumors within eight days. The authors hope to gain a clearer conception of the virus in their studies of the protein-free virus.

THE INFLUENCE OF FOOD STUFFS ON THE ACID BASE BALANCE OF CATTLE URINE. Frederick John Warth and Narayana Krishna Ayyar. Biochem. Jour., xxiv (1931), 6, p. 1595.

The urines from three types of fodders were examined by a method described by the authors. It was found that the urines from green fodders and cereal straws were alike in containing

excess of alkali and large amounts of carbon dioxid. In contrast to the above, the urines from over-ripe hays contained very little carbon dioxid and were neutral or acid. The urine of wheat straw is acid and forms an exception in its class. Progressive ripening of a fodder tends to increase the acidity of the urine. At the same time it was observed that the climatic conditions may materially modify the effect of the ripening. Hippuric acid is highly significant in the acid-base balance. The amount of carbon dioxid present in the urine is found to increase regularly with the excess of alkali.

THE EFFECT OF OILS ON GASTRIC SECRETION AND MOTILITY. W. Morrell Roberts. Quart. Jour. Med., xciv (1931), p. 133.

The efficiency of oils in inhibiting gastric secretion appears to ear some relationship to the degree of saturation of the constituent fatty acids, the less saturated oils being the more efficient. The relationship between efficiency on the one hand and specific gravity and viscosity on the other is of less significance. Of related substances only the free fatty acids show inhibitory properties. Oil diminishes the response of the stomach to the presence of food and other stimulatory substances within it but secretion due to histamine is not inhibited. Oil inhibits both the psychical and the chemical secretions. The action of oil is exerted mostly or entirely after it has left the stomach. The effect of oil on gastric motility is not secondary to its effect on secretion. The above facts are discussed and a theory advanced for the action of oil on gastric secretion and motility.

DEMONSTRATION OF A DOG MAINTAINED FOR SIXTEEN WEEKS SOLELY BY JEJUNAL ALIMENTATION IN THE PRESENCE OF LOSS OF GASTRIC JUICE. H. G. Scott, P. H. Holinger and A. C. Ivy. Proc. Soc. Exp. Biol. and Med., xxviii (1931), 6 p. 569.

The dog demonstrated has a pouch of the entire stomach with vagi intact and a jejunal fistula. The pylorus was cut across, the duodenal end closed and the gastric end brought to the outside. The jejunal fistula was made prior to this operation. The animal has been fed solely through the jejunal fistula for 18 weeks and has been losing all gastric juice (400-600 cc daily) for a period of 16 weeks. He has received nothing subcutaneously or intravenously except 1½ gr. ferric citrate during the last three weeks, every other day, because he became somewhat anemic. His

blood is now normal. The blood chlorid level can be varied at will by removing or adding sodium chlorid to his diet. The dog now weighs as much as he did prior to the operation (24 lbs.). At 2½ months a stomatitis with ulcers appeared, which disappeared as vitamins were added to the diet and the mouth washed daily with lemon-juice.

CARBOHYDRATE METABOLISM IN TUBERCULOSIS. I. E. Rabuchin. Abst. Arch. Path., xi (1931), 4, p. 641.

Both in experimental and in extensive pulmonary tuberculosis the carbohydrate metabolism is decreased. The impairment is due to a dysfunction of the liver and of the suprarenal system. In experimental tuberculosis in guinea pigs, there is a hypofunction of the pancreas. The condition of the carbohydrate metabolism justifies the therapeutic administration of insulin.

INCIDENCE OF BRUCELLA AGGLUTININS IN AN AVERAGE UNSELECTED INDIAN POPULATION. S. M. K. Mallick and M. L. Ahuja. Indian Jour. Med. Res., xviii (1931), 3, p. 983.

The sera of 382 cases (healthy and febrile) were tested for Brucella agglutinins, 247 being from healthy adults and 135 from febrile cases. Both the agglutinins of *Br. melitensis* and *Br. abortus* were tested for. Twenty-six samples from healthy cases gave a positive reaction in a dilution of 1:50, six by the use of *Br. melitensis* antigen and 20 when *Br. abortus* antigen was used. Twenty-five samples from febrile cases gave a positive reaction in a dilution of 1:50, eight with *Br. melitensis* antigen and 17 when *Br. abortus* antigen was used.

OCCURRENCE OF THE BOVINE TYPE TUBERCLE BACILLUS IN PULMONARY TUBERCULOSIS. Its role in pathogensis. Abst. Tubercle, xii (1931), 7, p. 328.

Bovine type bacilli are very rarely obtained from pulmonary lesions. During eight years, ten cases were discovered among 250 patients from whose sputum tubercle bacilli had been grown in culture. Most notable features of such cases were the amount of fibrotic changes present, the lack of evidence of much breaking down of lung tissue, and the absence of hemoptysis, the author pointing out that there appeared to be a better chance for most of these patients to overcome their systemic disturbance than would be the case with a similar group with human type infec-

tions. The author advances the idea that the bovine type of bacillus is not so capable of generalization or of producing visceral tuberculosis as the human type and also that the allergic reaction created by reinfection with the bovine type tubercle bacilli seems to be more proliferative and to show less tendency to breaking down of tissue. This he thinks may explain the absence of the bacilli in patients with pulmonary tuberculosis due to the bovine type. In such cases it is thought that the route of infection is through the tonsils or cervical glands.

THE CELL CONTENT OF DOG LYMPH. Florence W. Haynes and Madeleine E. Field. Amer. Jour. Physiol., xc ii (1931), 1, p. 52.

The number of cells in the lymph from different parts of the body as shown by those experiments agree with the data of other workers and show irregularities similar to theirs. The difference in cell content of lymph passing through areas with and without lymph-nodes is clearly demonstrated. The cervical lymph which has passed at least in part through lymph-glands was found to contain 2,800 to 68,600 white cells per cubic millimeter, whereas leg lymph, which had passed through few if any nodes, contained none to 2,500 cells. By a short period of massage of the cervical lymph-glands the white cell count could be greatly increased and gradually fell on the continued massage.

INTESTINAL ADENOMA IN SWINE. H. E. Biester and L. H. Schwarte. Amer. Jour. Path., vii (1931), 2, p. 175.

In experimental and field cases of infectious enteritis in which extensive mucosal destruction occurred, epithelial proliferation originating in the remaining islands of injured cells was noted. An extensive destruction of mucosal tissue in two severe advanced cases of enteritis was associated with a degeneration of the epithelium and the formation of adenomatous growths. The herd history suggests infectious enteritis as being the etiological factor. *Salmonella suipestifer* antigen was agglutinated in a titre of 1:25. The tumefied tissue consisted of glands lined by undifferentiated cells. The authors suggest a relationship between intestinal adenomatous proliferations and destructive processes followed by epithelial regeneration as against their interpretation as independent tumors arising from isolated or misplaced embryonic rudiments.

Regular Army

Captain Frank H. Woodruff is assigned to duty at Fort Bliss, Texas, effective upon completion of his present tour of foreign service in China.

Captain Joseph F. Crosby is relieved from duty at Fort Bliss, Tex., effective at such time as will enable him to proceed to San Francisco, Calif., and sail on transport scheduled to leave that port on or about September 10, 1931, for the Philippine Islands. Upon arrival at Manila he will report to the commanding general, Philippine Department, for assignment to duty with the Veterinary Corps at Tientsin, China.

First Lieutenant Stanley M. Nevin is relieved from assignment and duty at Fort Sam Houston, Texas, to take effect on or about June 15, 1931, and will proceed to Fort Riley, Kans., and report to the commanding general for duty.

Major Sherman R. Ingram is relieved from duty at Fort Logan, Colo., and assigned to Fort Huachuca, Ariz.

Captain Vincent B. Wright is relieved from duty at Fort Huachuca, Ariz., and assigned to Fort Bragg, N. C.

Captain Frank C. Hershberger is relieved from assignment and duty as a student at the Cavalry School, Fort Riley, Kans., effective upon completion of the present course of instruction, and will then report to the commanding general, Fort Riley, Kans., for duty.

Lt. Colonel Alfred L. Mason is relieved from his present assignment and duty at Fort Sam Houston, Tex., and will report to the commanding general, Eighth Corps Area, for duty with the Veterinary Corps at his headquarters.

Captain James A. McCallam is relieved from assignment and duty at the remount purchasing and breeding headquarters, Kansas City, Mo., and from additional duty with the Organized Reserves of the Seventh Corps Area, and will proceed to Fort Humphreys, Va., for duty at that post and for additional duty as attending veterinarian at Fort Washington, Md., and Fort Hunt, Va.

Captain John W. Miner is relieved from assignment and duty at Fort Leavenworth, Kans., will proceed to Kansas City, Mo., and report to the commanding officer, remount purchasing and breeding headquarters, for duty.

Veterinary Reserve Corps

New Acceptances

Merriman, Cecil Mathus . . . 1st Lt. . . 502 N. Vine St., Mt. Pulaski, Ill.
Emmerson, James Hiram . . . 1st Lt. . . 62 Chapman St., Wollaston, Mass.
Chastain, Walter Ralph . . . . 2nd Lt. . . 408-417 State Office Bldg., Columbia, S. C.
Puckett, Leo V. . . . . 2nd Lt. . . 215 S. 11th St., Mt. Vernon, Ill.

Promotions

Starkey, Jay Ralph . . . . . To 1st Lt. . . St. John St., Goshen, N. Y.

Doctor Topacio Returns to the Philippines

Dr. Teodulo Topacio, of the Philippine Islands, after a considerable stay in the United States, has returned home by way of Europe. Dr. Topacio received his veterinary degree from the University of the Philippines. He then spent some time in the Department of Agriculture laboratories in Manila. Later he spent one year at Washington State College in graduate work and obtained the degree Master of Science, his thesis being "Blood Groups in Animals." The following year was spent at the University of Pennsylvania Veterinary School, where he pursued the graduate course in veterinary medicine offered there, which includes work in pathology and bacteriology in the Medical School of Medicine, and for which he received a certificate.

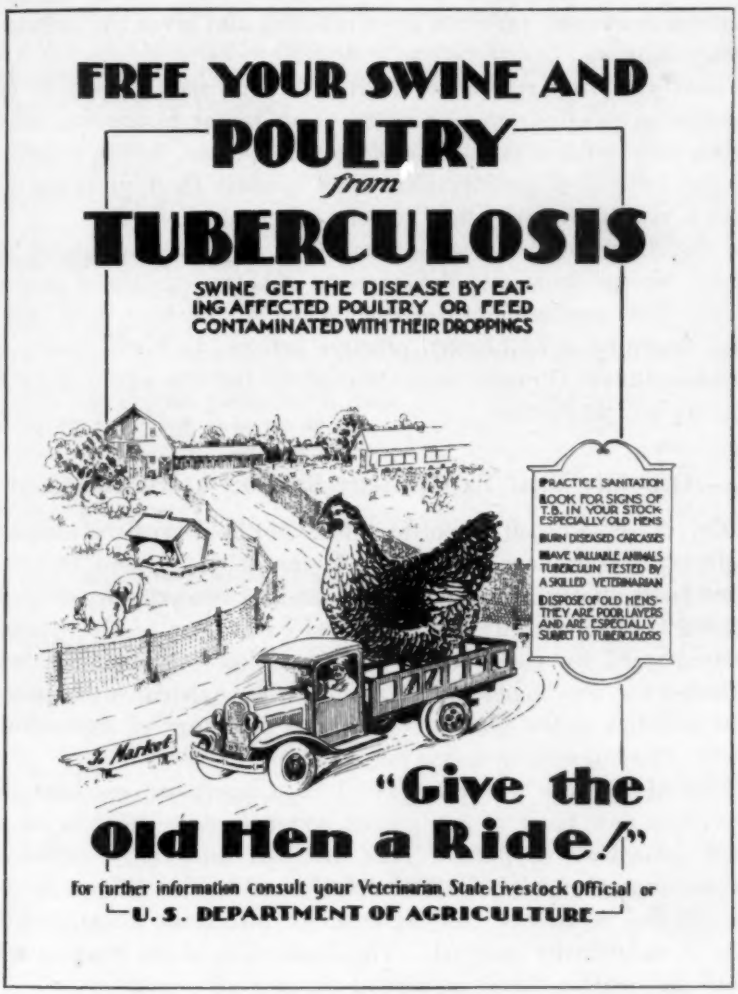
Dr. Topacio then went to Johns Hopkins Medical School, Baltimore, where he received the degree Doctor of Science in Hygiene, December 1, 1930. Because of advanced work in other institutions, Dr. Topacio was not required to take two years of course work at Johns Hopkins, but after special work in filtrable viruses for one year he was awarded his degree. This usually requires three years.

The experience of Dr. Topacio indicates the opportunity open to veterinarians so minded to study and also shows the importance with which some governmental institutions consider such advanced work, since Dr. Topacio was supported in his studies by the Philippine government. It is additional evidence of the close relations existing between the medical and veterinary professions.

Give the Old Hen a Ride

Showing a giant hen in a motor truck speeding to market, a new poster of the U. S. Department of Agriculture advises poultrymen to "Give the Old Hen a Ride," at the same time

giving the pullet a chance. The poster points out that disposing of old hens tends to keep the poultry flock free from tuberculosis, a disease that principally affects fowls more than 1½ years old.



New poster for use in campaign against avian tuberculosis.

By disposing of hens at that age, the appearance to tuberculosis in flocks is largely prevented and in addition there is less likelihood of the same disease appearing among swine, which often become infected by eating the carcasses of tuberculous hens or feed contaminated with their droppings.

A further benefit, according to the Department of Agriculture specialists, resulting from the disposal of old hens, is the improvement in egg production in farm flocks. The marketing of old birds at the end of their first laying year, after which egg production decreases, prevents overcrowding and gives the pullets a better chance.

That the Department of Agriculture officials recognize the practicing veterinarian as an important factor in the control of avian tuberculosis is evidenced by the poster, which suggests to the owners of poultry that they consult their veterinarian, with a view to having their poultry tuberculin tested.

Officials of poultry associations have requested copies of the poster for distribution in the interests of better quality in poultry meat. The marketing of fowls while tender, healthy, and otherwise desirable is of benefit, officials believe, to both producers and consumers through improvement in the average quality of poultry on the market.

Officials Visit Experiment Station at Bethesda

Dr. W. E. Cotton, superintendent of the Bureau of Animal Industry Experiment Station at Bethesda, Md., and his staff were hosts, Saturday afternoon, June 20, to approximately 50 administrative officials and technical employees of the Bureau accompanied by members of their families. Opportunity was afforded for the inspection of laboratories, experiment animals, and facilities of the Station. Assistant Secretary of Agriculture R. W. Dunlap was an honor guest.

Special interest was manifested in a pipetting machine, an ingenious new piece of equipment which rapidly delivers measured quantities of fluid. This machine materially expedites bacteriological work. Use of colored media in differentiating the several strains of the organism of infectious abortion also was of noteworthy interest. The inspection of the Station was concluded with a luncheon served picnic style.

Deer found dead in Pennsylvania forests in recent years were thought to have died from laurel poisoning, but no direct evidence as to the exact cause of death could be found, and experiments by Pennsylvania State officials in feeding deer exclusively on laurel leaves did not cause poisoning except when they were forced to eat large quantities of mountain-laurel.

AMERICAN VETERINARY MEDICAL ASSOCIATION

Financial Report—Dr. M. Jacob, Treasurer

For the year ending December 31, 1930

Cash in bank, December 31, 1929 (checking accounts)		$ 4,429.96
Receipts, January 1, 1930, to December 31, 1930:		
Received from the Secretary	$35,341.79	
Interest on time deposits	1,528.88	
Proceeds from sale of Canadian bonds (par)	7,000.00	
Interest on bonds	382.50	
Total receipts		$44,253.17
		$48,683.13
Expenditures for the year 1930		36,355.57
Balance		$12,327.56
Placed on time deposit during year		12,343.29
Cash in bank, December 31, 1930 (checking accounts)—overdraft		$ 15.73
Revolving fund in hands of Dr. Hoskins		500.00
Cash on time deposit, December 31, 1929	$22,794.38	
Added during period (as shown above)	12,343.29	
		$35,137.67
Total cash resources		$35,621.94
Bonds (purchase price)		8,674.64
Due from Salmon Memorial Fund		150.00
Total assets, December 31, 1930		$44,446.58
Total assets, December 31, 1929		43,205.29
Increase for the period		$ 1,241.29

Distribution of Assets

A. V. M. A. Fund

Cash (overdraft in checking account)	$ 409.62
Bonds (cost)	5,821.94
Loan to Salmon Memorial Fund	150.00
Total	$ 5,562.32

Journal Fund

Cash	$36,031.56
Bonds (cost)	2,852.70
Total	$38,884.26

Salmon Memorial Fund

First Mortgage bonds (5½%)	$ 5,500.00
U. S. Liberty Bond (par)	500.00
Time deposits	385.66
Total	$ 6,385.66

COMMENCEMENTS

ALABAMA POLYTECHNIC INSTITUTE

The commencement exercises of the Alabama Polytechnic Institute were held May 19, 1931. In the College of Veterinary Medicine the following graduates received the degree of Doctor of Veterinary Medicine:

Van F. Bess
A. R. Griffith
V. D. McCreary

KANSAS STATE COLLEGE

The sixty-eighth annual commencement exercises of the Kansas State College were held at Manhattan, May 28, 1931. In the Division of Veterinary Medicine the degree of Doctor of Veterinary Medicine was conferred upon the following:

John Bertram Cheshire
Walter Geurkink
Robert Bruce Helming
Elmer David Johnston
Wayne Otho Kester
Forrest Coniver Love
Andrew Lafayette McBride
Carl Jacob Majerus
Clayton John Price
Willet Jesse Price
Don Harvey Spangler
Dale Suplee
Elliott Rodney Trull

The following graduates received commissions as second lieutenants in the Officers' Reserve Corps of the United States Army:

Elmer David Johnston
Forrest Coniver Love
Clayton John Price
Willet Jesse Price
Don Harvey Spangler
Dale Suplee
Elliott Rodney Trull

Andrew Lafayette McBride was the honor student in the Division of Veterinary Medicine.

Sophomore honors were awarded to Richard Duncan Turk and Wm. H. Lindley.

A. AND M. COLLEGE OF TEXAS

At the commencement exercises of the A. and M. College of Texas, held May 30, 1931, the following graduates received the degree of Doctor of Veterinary Medicine:

F. M. Burkey
W. C. Butler
R. T. Dickinson
Chas. W. Neal
R. E. Starnes
C. C. Young

All of the graduates took the Texas State Board examination, June 1, and were given licenses to practice.

COLORADO AGRICULTURAL COLLEGE

At the commencement exercises of the Colorado Agricultural College held in the Men's Gymnasium Building, June 4, 1931, there were thirteen graduates in veterinary medicine:

Clifford W. Barber
Gordon W. Barr
Carl B. Bills
Douglas Brown
Edgar F. Ebert
Kenneth L. Green
Henry B. Lake
Theodore H. Leenerts
Peter E. Madsen
Theodore R. Myers
Roy C. Park
John H. Williams
Richard G. Yule

The commencement address, "Life's Challenge and Opportunity," was given by Rev. David Carson Bayless, of the Humphreys Foundation, Denver, Colo.

On May 27 and 28, the Colorado Veterinary Medical Association held its semi-annual meeting at the Agricultural College. In connection with the banquet, which was held on the evening of the first day, the Alpha Psi cup was presented to Mr. Lester Heath. This cup is presented each year to the student receiving the highest grades in the sophomore year. Dr. C. E. Salsbery presented the Jensen-Salsbery award for the best work in surgery. The first award of $15.00 went to Richard G. Yule, a senior, and the second award of $10.00 went to Curtis Hagler, a junior.

Among the visitors, in addition to Dr. Salsbery, were Dr. E. R. Frank, Manhattan, Kans., and Dr. Frank Breed, of Lincoln, Nebr.

OHIO STATE UNIVERSITY

The fifty-fourth annual commencement exercises of the Ohio State University were held in the Ohio Stadium, Monday, June 8, 1931. The commencement address was delivered by Julius Howland Barnes, LL.D., D. Bus. Adm., chairman of the Board of the United States Chamber of Commerce.

The College of Veterinary Medicine presented the following candidates for the degree of Doctor of Veterinary Medicine:

Howard M. Aitken
Robert Paris Armstrong
Leonard Perry Bailey
Kenneth Leland Benner
Paul Clifford Bennett
William Clinton Bowen
James Thomas Burriss
Kenneth Kent Burris
Gordon Tobias Castor
Paul Markel Cellar
Glen Garold Crosbie
Walter Harmon Fenner
John Fremont McClure
Henry Strom Magnus
Gerard Boylan Merrick
Granville John Miars
William Karl Mueller
John Joseph Murphy
Thomas Presow Nankervis
Charles Robert Pastors
James Ralph Peters
John Lyle Putnam
Leonard Robert Richardson
James Lawrence Smith

Walter C. Ferrall
Charles Christian Gluhm
Amor Edward Hancock
Leo Henry Hartman
Carl Sewell States
Fritz Volkmar
Raymond Dean Wenger
Raymond Francis Witt

The degrees were conferred by President George W. Rightmire, and diplomas awarded following the formal presentation of the candidates by Dean Oscar V. Brumley.

Fifteen members of the graduating class immediately filed applications for membership in the A. V. M. A.

CORNELL UNIVERSITY

The annual commencement exercises at Cornell University were held June 15, 1931. The degree of Doctor of Veterinary Medicine was conferred upon the following:

Alfred M. Beers
Frederick C. Cairns
Hugh S. Cameron
Lyle S. Compton
Clyde E. Constable
James C. Crandall
Douglas B. Crane
Simon P. Dansky
Carlton C. Ellis
Sebastian B. Fischer
Arthur W. Fredericks
Theodore W. Goers
Harry E. Hansen
William E. Jennings
Grant S. Kaley
Abraham Kleinfeld
Raymond C. Klussendorf
John W. Knapp
Asa F. Legg
Paul D. Marvin
Irwin M. Moulthrop
Douglas M. Overacker
Alexander L. Raebone
William S. Shaw
Albert M. Snelling
George J. Strang
Benjamin W. Suydam, Jr.
Quinton L. Todd
Lawrence T. Waitz
Nathan Wernicoff
Elmer A. Woelffer

The following prizes were awarded for the academic year 1930-1931:

The Horace K. White Prizes (Meritorious Students):
First Prize.....Abraham Herman Kleinfeld
Second Prize.....Paul Dana Marvin

The Jane Miller Prizes (Veterinary Physiology):
First Prize.....John Chester Stevenson
Second Prize.....Burton Fuller Judson

The James Gordon Bennett Prizes (Surgical Clinics):
First Prize.....Harry Edward Hansen
Second Prize.....Simon Paul Dansky

The Anne Besse Prize (Veterinary Medicine).....Elmer August Woelffer

The Charles Gross Bondy Prizes (Small-Animal Clinics):
First Prize.....Lyle S. Compton
Second Prize.....Douglas Belden Crane

The Merry Prizes (Anatomy):
First Prize.....Henry Elmer O'Neil
Second Prize.....Edward Clark George

IOWA STATE COLLEGE

Commencement exercises at Iowa State College were held on Monday, June 15, 1931. On that occasion the degree of Doctor of Veterinary Medicine was conferred on 38 candidates, the largest class to be graduated from Iowa State College. Those graduated are as follows:

Ralph H. Bergman
Lloyd C. Blank
Kenneth Bogaard
Marion H. Carter
Warnie R. Collins
Kenneth H. Fritts
Ellis H. Gloss
A. Holland Groth
Vincent Held
Gregory Jennings
Lloyd D. Jones
Lester W. Larson
Clarence Lekwa
Bryan F. Lott
Duane Lyddy
Paul J. Meginnis
Harold L. Morrison
E. Michael Noethe
Russell O. Nye
Wm. T. Oglesby
Carl Olson, Jr.
Ernest W. Paulsen
W. Boyd Penrose
Charles K. Pfaff
Howard D. Perry
Bennett J. Porter
Raymond F. Rasmussen
Henry Schwermann
George A. Snyder
Maynard L. Spear
Laurence Sundberg
Elmer Sundquist
Harold L. Strandberg
Clarence L. Taylor
Chun Lin Teng
Henry G. Voetberg
Ernest F. Waller
Doran Yegian

George A. Snyder led the group in scholarship and accordingly was awarded the George Judisch Prize, consisting of initiation fee and membership dues in the A. V. M. A.

Two new prizes were awarded for the first time this year for the best students in clinical medicine. The first prize of $15.00 was awarded to A. H. Groth, and the second one of $10.00 was won by Wm. T. Oglesby. These prizes were given by Dr. G. C. Graham, of the class of 1908 and a classmate of Dr. Bemis.

UNIVERSITY OF PENNSYLVANIA

At the annual commencement exercises of the University of Pennsylvania, held June 17, 1931, the degree of Doctor of Veterinary Medicine was conferred upon the following:

Reubin Yechiel Berenstein
Robert Arthur Boyce, Jr.
Ralph Corey Briggs
John Malcolm Coffin
George Eugene Cohan
Alfred Everett Coombs
Edwin Carl Frederick Enge
Alexander Rae Evans, Jr.
Herbert Frederick Harms, Jr.
Roy Donald Hoffman
Ralph Frederick Kornman
Lawrence John Manogue
Todd Oulette Munson
Carl Louis Schulster
Hermann Rudolph Seibold
Edward Barnwell Smith
Quinton Wendell Sparks
Dinny Lup Tong
Willard Sherman Young, Jr.

The J. B. Lippincott Prize of $100.00, for the highest general average for the entire four years of the course, and the T. E.

Munce Prize of $25.00, for the highest general average in the courses in Animal Husbandry, were awarded to George E. Cohan.

The Jeannette Blair Prize of $50.00, for the best work done in the Small-Animal Clinic, was awarded to Herman R. Seibold, who also won the Leonard Pearson Prize of $50.00. This prize is awarded to the member of the senior class who has shown, in the opinion of the veterinary faculty, by his scholarship, breadth of interest, personality and high character, combined with his ability to speak and write correct English, that he is most capable of dignifying and advancing veterinary science in research, in practice, in education and in its relation to civilization.

UNIVERSITY OF GEORGIA

At the 130th commencement exercises of the State College of Agriculture and Mechanical Arts, University of Georgia, held June 17, 1931, the degree Doctor of Veterinary Medicine was conferred upon the following:

George Thomas Adair
Frank S. Carr
John Samuel Lide
Jim Hill McClung
Russell E. Whitcomb
James Mack Wooldridge

William Dana Hiscock and Francis L. Tarver expect to complete the work for their degrees during the summer session.

MICHIGAN STATE COLLEGE

At the seventy-third annual commencement exercises of the Michigan State College, held June 21, 1931, the following graduates in the Division of Veterinary Science received the degree of Doctor of Veterinary Medicine:

Bernard Victor Alfredson
James Horace Campbell
Leo Philip Doyle
Ray Henry Fish
Eugene Ernst Hamann
Stanley Martinkewz
James Philip Torrey

The degree of Master of Science was conferred upon Dr. Claude Reading (Mich. '27).

The Michigan State Veterinary Medical Association prize of $25.00 was awarded to Bernard Victor Alfredson, whose senior record was the highest for the class. James Horace Campbell and Stanley Martinkewz received commissions as second lieutenants in the Reserve Corps of the United States Army.

Since the 1930 commencement, the degree of Doctor of Veterinary Medicine was conferred upon Connor D. Smith.

CORNELL CONFERENCE FOR VETERINARIANS

The twenty-third annual Conference for Veterinarians at Cornell University was held in James Law Hall, January 15-16, 1931. Dr. H. C. Stephenson presided at the first session. Dr. P. A. Fish spoke on "The Rise of the Veterinary Profession." He said that cattle were imported to America by Columbus, and horses by other Spaniards. Tuberculosis early developed, and contagious pleuro-pneumonia and tick fever did an immense damage. These diseases led to the organization of the Bureau of Animal Industry, with Dr. D. E. Salmon at the head. Quarantine was established against contagious pleuro-pneumonia. The quarantine was established for the eastern coast and was under control of the Treasury Department. This activity was later transferred to the Bureau of Animal Industry. Such efforts led to the eradication of contagious pleuro-pneumonia in 1892. Federal meat inspection was established to remove a European embargo. It was later extended to cover the whole country. Veterinary schools were established between 1860-1870. The education of practitioners had become necessary in order to fight sporadic diseases. The laboratory increased in importance with the discovery of tuberculin, mallein, the agglutination test for Bang's disease, and the color test for mastitis. Much progress has been made in tick and tuberculosis eradication. Health and prosperity are at stake and competent veterinary service has become a factor which can no longer be ignored. This service is not a branch of agriculture. The diagnosis and treatment of animal diseases requires a special training and experience.

Dr. W. T. Miller spoke of the important work, "Blood Volume Determination in Cattle," which he has carried out. He had made 40 determinations on 12 animals. The volume varied from 27.5 to 30 cc per pound of body weight. A 700-pound cow would have a blood volume of 25,000 cc. There is an increase in volume, up to a certain point, with increase in weight. Pregnancy

increases the volume up to parturition and in two to three weeks after parturition there is a decrease of 2500 to 3000 cc. The blood in the body of a cow would be around 1/12 of the body weight. The author states that his results are relative and the report of a preliminary nature. He used the vital red method and states the possibility that the volumes determined may be true plasma volume rather than blood volume.

Dr. L. R. Vawter, of the University of Nevada, has been spending a year at Cornell doing graduate work. He presented a paper, "Bacillary Hemoglobinuria of Cattle." Dr. Vawter described the disease as acute, infectious and non-contagious. It is a western disease and occurs on poorly drained, swampy or exceedingly irrigated pasture. The time is the summer and early autumn months. It is the occasional rather than the spectacular losses which make the aggregate. The disease follows a sudden rapid course, with a temperature of 104-107° C. Mortality is 95 per cent in 24 to 36 hours. There is pronounced hemoglobinuria with hemolysis of 50 to 75 per cent of the red cells in 24 hours. There is a marked disturbance in the oxidative processes. Anthrax, anaplasmosis, pyelo-nephritis, chronic cystic hemoglobinuria and bracken poisoning must be considered in differential diagnosis. In the treatment early purges should be avoided; no food, but plenty of water, should be given until the hemoglobinuria passes off. Subcutaneous injection of 10 to 20 cc of a bacterin, made up of dead organisms and toxins, is part of the treatment.

Dr. S. A. Adsell, Department of Animal Husbandry, College of Agriculture at Cornell, read a paper, "Recent Developments in the Field of Sex Hormones."

Dr. W. A. Hagan presented the last paper of the session, "The No-Lesion-Case Problem in the Tuberculosis Eradication Campaign." Dr. Hagan pointed out that the error in 75,000,000 tuberculin tests to June 3, 1930, was probably less than 1 per cent. The no-visible-lesion cases amount to about 11 per cent of the reactors, but is considerably higher in some localities. Such cases are not necessarily non-tuberculous. Gross lesions may not have developed and may not be seen if they have. Non-tuberculous animals sometimes react to tuberculin. Some are hypersusceptible to the constituents of the broth in tuberculin. The probable cause of the greater number of no-visible-lesion cases is hypersensitization by non-pathogenic acid-fast organisms which are abundant in nature. It is certain that cattle

and other animals are sensitized to tuberculin by the injection of such organisms. In nature they invade the digestive tract and skin. Evidence that such invasion causes the tuberculin reaction is lacking.

Dr. M. G. Fincher presided over the afternoon session of the first day. Dr. W. L. Williams gave an extremely interesting illustrated talk, "Notes on the Animal Industry of Hawaii." He had just recently returned from a year in Hawaii and gave us the full benefit of his experience and observations.

Dr. H. Lothe, of Waukesha, Wisconsin, gave the next paper. His subject, "Some Clinical Observations on the Agglutination Test for *B. Abortus* as a Means of Controlling Abortion and Other Genital Diseases of Cows," was an avenue through which the speaker was able to pass on to his audience the benefits of a rich experience in the use and interpretation of the test. Dr. Lothe emphasized the value of the test to the practitioner and showed that practice justified its application.

Dr. H. L. Gilman next reported on "Further Studies on the Relation of the Milk Titre to the Elimination of *Bact. abortus* from the Udder." His findings indicate that the organism was recovered from 58.6 per cent of the animals showing a positive blood titre. The organism was recovered but once from a quarter showing agglutinins at less than positive at 1:80, and from quarters positive at 1:80 or higher it was recovered in 60.4 per cent of the samples. Evidence points to the fact that milk agglutinins are produced locally in each quarter of the udder. It was indicated that the dilution 1:80 might be taken as a dividing line between negative and positive cases as in the blood reaction. The reaction at a dilution of 1:80 or above indicated that 75 per cent of 104 animals examined had infected udders. A considerable degree of relationship between the agglutination titre of milk and *Bact. abortus* in it was indicated.

Dr. John McAuliff contributed the results of valuable experience in his paper, "Bang Abortion Disease from the Practitioner's Standpoint." Dr. R. R. Birch opened the discussion on the last three papers and a great deal of interest was manifested by the number of practitioners participating.

Dr. R. L. Conklin, MacDonald College, Quebec, Canada, next discussed his findings in "The Clinical, Bacteriological and Chemical Study of the Pregnant Uterus." Some very definite differences are manifest in the bacteriology and chemistry of the amniotic fluid from normal and abnormal uteri. Bacterial

flora was found to be present in 71 of 80 pregnant bovine uteri studied. The type of organism seemed to be governed by the pH, density and nitrogen content of the amniotic fluid.

Dr. P. A. Fish presided at the evening session. Professor C. L. Durham gave the address of welcome on behalf of Cornell University. Professor F. K. Richtmyer gave a lecture on the X-ray. Prof. Richtmyer is a noted authority in this field. He used X-ray apparatus of various types to illustrate his talk. His illustrations and explanations made the X-ray a very interesting subject to a thoroughly attentive audience. At the close of the lecture we adjourned to James Law Hall, where a smoker and get-together concluded the program for the day.

Dr. J. W. Benner presided at the morning session of the second day. Dr. H. C. Stephenson gave a talk on "Nembutal 844." He showed a dog and cat under the influence of the preparation and pointed out some of its advantages over the older anesthetics. He was careful to indicate that his use of the preparation was yet in the experimental stage.

Dr. H. J. Milks gave the next paper. He took as his subject "Some Surgical Diseases of the Abdomen." This paper was especially profitable, for every disease touched upon by the speaker was a part of his rich and varied experience in the surgery of small animals.

Dr. Oskar Seifried, Rockefeller Institute, Princeton, N. J., illustrated his lecture on "Borna Disease" with a film. The film was a very graphic portrayal of the symptoms of the disturbance as it affects different animals. The speaker's discussion of his topic was a valuable contribution to the program.

Dr. J. N. Frost talked next on "Diseases of the Synovial Sheaths and Bursae." He illustrated his talk with specimens of normal and diseased limbs. This demonstration brought out a great deal of discussion.

Dr. John R. Mohler gave the next paper, "Some Activities of the Bureau of Animal Industry." It is superfluous to say that Dr. Mohler instructed and interested the members of the Conference. He paid just tribute to the names of V. A. Moore and P. A. Fish when he placed them among the few outstanding for their contributions to the progress of the veterinary profession. We at Cornell shall always remember and appreciate that tribute.

Dr. E. Sunderville presided at the afternoon session. Dr. D. H. Udall gave a paper, "The Diagnosis and Control of Mastitis," which was accompanied by slides illustrating the diagnosis

and results in several cases of mastitis. The value of the different means of diagnosis and methods of control were fully discussed by the speaker. "Field Observations on Mastitis" was presented by Drs. H. B. Switzer and C. W. Gates, Rouses Point, N. Y. These two papers brought out a great deal of helpful discussion. Prof. C. N. Stark, of the Dairy Department, Cornell University, gave some valuable data on "The Characteristics of Organisms Causing Ropy Milk and the Importance of Feeds as Their Source."

The dinner session was held at the Masonic Temple, on Friday evening. This particular dinner, a closing event of each conference, was to be the last conference banquet to be graced by the presence of Dr. V. A. Moore and Dean P. A. Fish. For twenty-three years, Dr. Moore and Dr. Fish had labored to make the annual Conference for Veterinarians an event of first importance among the activities of the College. The increasingly large number of veterinarians in attendance at each meeting amply demonstrated that their efforts had met with marked success. Approximately 250-300 places were filled at the 23rd banquet.

A distinguished guest was Dr. John R. Mohler, Chief of the U. S. Bureau of Animal Industry. Dr. P. A. Fish presided. Dr. H. H. Horner, Director of State College Education, State Educational Department of Albany, N. Y., in an excellent address, clearly indicated that he is familiar with the progress made by the veterinary profession. Veterinary education, he pointed out, has had a tendency to become more and more scientific and he praised the efforts of Dr. Law, Dr. Moore and Dr. Fish for the part that they had played in bringing this about. Veterinary medicine has in no sense decreased in importance with the decline in the horse population. There exists no wide gap between veterinary medicine and human medicine and public health is in a large measure dependent upon the services of the veterinary profession.

Dr. L. A. Maynard, Department of Animal Husbandry, College of Agriculture, Cornell University, gave a paper dealing with the subject of the fat content in milk. Dr. Maynard and his co-workers have found that exercise tends to increase the fat content. Low stable temperatures, within physiological limits, also tend to increase the content of fat in the milk. He showed that no system of feeding will permanently increase the

fat content; that feed primarily affects the yield and not the fat content percentage.

Dr. V. A. Moore emphasized the opportunity and the responsibility which the veterinarian has in relation to the production of safe milk. The veterinarian, he said, is responsible not alone to the public at large, but to the farmers and the members of his family as well who consume a large portion of the milk produced.

The part of the program of the evening which was of especial interest and significance was the presentation of a diploma, by Dr. John R. Mohler to Dr. V. A. Moore, which signified the membership of Dr. Moore in the Royal Society of Veterinary Surgeons of England. Dr. Mohler and Dr. Moore were the two veterinarians from the United States elected to this Society at the 1930 International Veterinary Congress. Dr. Moore was not in attendance at the Congress. In a brief response, Dr. Moore said that he was very grateful that so signal an honor had been paid to him. He expressed himself as holding in the highest esteem the president of the Society, Sir John M'Faydean.

This dinner closed the best of all the Conferences held at Cornell. Dr. Moore gave himself without stint to the twenty-one under his guidance. Dr. Fish continued the standard of excellence in the last two. They are no longer here to guide and advise but they have left such an impetus to success that we plan and hope always to make the next always a little better than the one preceding.

C. E. Hayden
Jesse Sampson, *Reporters.*

EASTERN STATES LABORATORY WORKERS' CONFERENCE

The fourth annual conference of the Poultry Disease Laboratory Workers was held at the Department of Veterinary Science, Massachusetts State College, Amherst, Mass., May 4-5-6-7, 1931. Twenty-eight members were in attendance, representing the following states and provinces: Connecticut, Delaware, Maine, Maryland, Massachusetts, North Carolina, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, Vermont, West Virginia, Ontario and Quebec.

The objective of these conferences is to establish more uniform methods employed in pullorum disease diagnosis and eradication

and to investigate those problems which are encountered in the diagnostic and eradication work.

The different members contributed data bearing upon the following subjects: (1) antigen (media, age limits, hydrogen-ion concentration, comparison of antigens and strains selected); (2) temperature and length of incubation period for agglutination; (3) hemolyzed samples; (4) "jelled" samples; (5) diagnostic dilution; (6) earliest age of birds for testing; (7) short-interval testing; (8) necropsy service as a follow-up measure in doubtful flocks; (9) soil infection; (10) fowl other than chickens; (11) whole-blood test; (12) postmortem technic and (13) comparative readings.

The report by the Committee on Standard Methods of Diagnosis was unanimously approved by the conference.

Summary of 1930-1931 Testing Data Submitted by the Laboratories

Laboratory	Samples Tested	Percentage of Infection	Number of Flocks	Number Positive
Connecticut	161,734	1.45	178	55
Cornell	46,420	8.22	97	85
Delaware	89,409	7.92	328	299
Farmingdale, L. I.	11,748	3.16	18	14
Maine	117,046	0.42	239	35
Maryland	16,297	17.13	30	30
Massachusetts	402,983	1.47	447	119
New Hampshire	164,510	0.92	172	36
Ontario Agricultural and Veterinary Colleges	112,635	5.13	399	329
Pennsylvania	238,229	6.69	434	384
Rhode Island	20,887	5.24	33	31
Vermont	27,584	0.93	72	24
Virginia	135,282	11.41	607	559
West Virginia	38,000	9.16	290	207

Included also in the program were:

"Recent Findings in Coccidiosis of Chickens," Dr. E. E. Tyzzer and Dr. E. Elizabeth Jones, Harvard Medical School.

"Fowl Paralysis," Dr. C. L. Martin, University of New Hampshire, and Dr. Oskar Seifried, Rockefeller Institute for Medical Research.

"Infectious Laryngo-tracheitis," Dr. J. R. Beach, University of California (on leave of absence at Rockefeller Institute), Dr. Oskar Seifried, and Dr. Charles S. Gibbs, Massachusetts Agricultural Experiment Station.

Correspondence relative to the 1932 meeting, which will be held at Cornell University, should be addressed to Dr. E. L. Brunett, Ithaca, N. Y.

HUDSON VALLEY VETERINARY MEDICAL SOCIETY

The regular quarterly meeting of the Hudson Valley Veterinary Medical Society took place at Catskill, N. Y., on May 13, 1931. About forty-five, including ladies and guests, sat down to a most excellent luncheon at the Saulpaugh Hotel, after which the party adjourned to the hospital of Dr. L. L. Parker, where the formal program followed, President Eichhorn presiding.

The Society was honored by the presence of the Chief Veterinary Officer of the Army Corps of Cuba, Colonel Luis I. Baltran, who briefly addressed the gathering.

After a brief business session, Dr. R. H. Spaulding, of White Plains, N. Y., was introduced by President Eichhorn and gave an excellent address on "Nephritis in the Dog," discussing causes, symptoms and treatment, in a very comprehensive manner. Copies of the address have been prepared for distribution to members of the Society and a limited number are available for others who may be interested.

After some discussion of Dr. Spaulding's paper, the President referred to some experiences in connection with symptoms appearing in horses used in the preparation of serum for the treatment of infantile paralysis. He stated that further studies were being made and would be available some time in the future.

Dr. Norman J. Pyle, Pearl River, N. Y., then gave an interesting demonstration of blood transfusion in the dog and postmortem methods in chickens. He answered numerous questions in relation to poultry diseases and explained many points in relation to troubles in small animals that interested those present.

After some further discussion, the Society accepted the invitation of Dr. Wright J. Smith, of Kingston, to hold the next meeting at Kenosha Lake, Ulster County, N. Y., August 12, 1931.

J. G. Wills, *Secretary.*

ILLINOIS STATE VETERINARY MEDICAL ASSOCIATION

The forty-ninth annual meeting of the Illinois State Veterinary Medical Association was held at Springfield, May 27-28, 1931. About 250 veterinarians were in attendance and the meeting was judged a complete success. The sessions were well attended and considerable interest was manifested in the program. The number of ladies and visitors who were in attendance was very gratifying to those in charge of the program. The golf tournament

held on the afternoon preceding the opening of the meeting was a big success.

The most important feature of the meeting was the action taken authorizing the Executive Board to employ an executive secretary who shall devote all or part of his time, to be decided later, in promoting the material welfare of members of the Association. Preliminary steps were taken to make this arrangement a permanent feature by introducing appropriate amendments to the Constitution and By-laws. The essential change will be an increase in the annual dues to finance the undertaking. It is estimated that it will be necessary to raise the dues to $15.00 annually, in order to be able to defray the expenses incidental to employing an executive secretary and to cover the expense of promotional activities, such as may be directed by the Executive Board.

The number of active members was increased by 77, and about 100 members have already paid their dues and assessments in full for the current year.

The Association was fortunate in being able to have three visiting veterinarians contribute to the program. These were Major G. W. Dunkin, of Mill Hill, England; Dr. Maurice C. Hall, Washington, D. C.; and Dr. Louis D. Mersch, of Des Moines, Iowa.

Officers elected for the ensuing year are: President, Dr. L. N. Morin, Clinton; vice-president, Dr. A. E. Dickerson, Springfield; secretary-treasurer, Dr. J. V. Lacroix, Evanston; members of the Executive Board, Dr. A. C. Bolle, Petersburg; and Dr. C. C. Hastings, of Williamsville.

J. V. Lacroix, *Secretary.*

SOUTHEASTERN MICHIGAN VETERINARY MEDICAL ASSOCIATION

The annual dinner meeting of the Southeastern Michigan Veterinary Medical Association was held at the Bean Pot, Detroit, June 1, 1931.

On this occasion, the Association had the great pleasure of entertaining Major G. W. Dunkin, of Mill Hill, England. Following the dinner, Major Dunkin delivered a splendid address on the investigations of canine distemper conducted by the National Institute for Medical Research, at the Farm Laboratories, Mill Hill, England. Many of the veterinarians were accompanied by

their wives, and the officers of the Detroit Kennel Club were invited to attend the meeting as the guests of the Association.

It is needless to say that everybody enjoyed Major Dunkin's address. He thoroughly covered the investigations of canine distemper from their inception down to date. Although every veterinarian present was more or less familiar with this work, through reports published from time to time, everyone felt keenly grateful for the opportunity of hearing about these researches direct from Major Dunkin.

Major Dunkin spent approximately four days in Detroit, and a number of the local veterinarians had an opportunity to entertain Major Dunkin and become better acquainted with him. An opportunity was afforded Major Dunkin to visit the biological farm of Parke, Davis and Company, at Rochester, Mich., prior to the meeting on June 1. The following day he was entertained at a luncheon at the Detroit Yacht Club, as the guest of Parke, Davis and Company.

SOUTHERN KANSAS VETERINARY MEDICAL SOCIETY

A meeting of the Southern Kansas Veterinary Medical Society was held at the Hotel Lassen, Wichita, Kans., June 8, 1931. The meeting was held during the afternoon, from 2:30 until 5:30.

A very interesting paper on "Digestive Disturbances in the Bovine" was given by Dr. E. J. Frick, of Kansas State College. This paper was well worth the time of every veterinarian in attendance because it was given from the viewpoint of the practitioner. Dr. Frick also spoke on various phases of small-animal practice and the use of various anesthetics. He also discussed the use of calcium chlorid solution in the treatment of eclampsia in bitches.

Dr. J. W. Lumb, of the Extension Department, Kansas State College, gave a short talk on a pamphlet which he is preparing for educational purposes to be put on the Santa Fe beef train which will visit 45 counties in Kansas, during the month of August.

Following the meeting a dinner was served in the dining-room of the Hotel, with wives and friends of the veterinarians participating.

M. L. Dietrich, *Secretary.*

NECROLOGY

HENRY WILLIAM FELDWISCH

Dr. Henry W. Feldwisch, of Piqua, Ohio, died at the White Cross Hospital, Columbus, Ohio, June 1, 1931. The immediate cause of death was a pulmonary embolism and acute heart disease.

Born October 20, 1890, near New Knoxville, Ohio, Dr. Feldwisch was graduated from Ohio State University in 1913 and served as a field veterinarian with the Division of Live Stock Industry, Ohio Department of Agriculture, until a little over a year ago, when he resigned to go into private practice. He located at Coshocton, Ohio, later removing to Piqua.

Dr. Feldwisch joined the A. V. M. A. in 1920. He was a member of the Ohio State Veterinary Association, and was affiliated with Humboldt Lodge, F. & A. M. He is survived by his widow, one son, his father, three brothers and three sisters.

ROBERT EMMETT NESBITT

Dr. R. E. Nesbitt, of Clinton, Ill., died June 17, 1931, following a skull fracture received in an automobile accident the day before, two miles north of Wapella, Ill., while en route to Kankakee on official business.

Born at Maroa, Illinois, July 3, 1872, Dr. Nesbitt received his veterinary training at the Chicago Veterinary College and was graduated in 1902. He started practicing at Maroa, Ill., and remained there until 1907, when he removed to Lincoln, Ill. He remained there until 1912, when he went to Clinton. From 1924 to 1928 he served as coroner of Dewitt County. He then received the appointment of Logan County Veterinarian, and for the past year was an assistant state veterinarian.

Dr. Nesbitt joined the A. V. M. A. in 1917. He is survived by his widow (née Nellie Hendrix), one son, two brothers and two sisters. Both brothers are veterinarins, Dr. A. S. Nesbitt (Chi. '94), of Decatur, Ill., and Dr. W. V. Nesbitt (Chi. '06), of Lincoln, Ill.

J. C. HARLOFF

Dr. J. C. Harloff, of Stamford, N. Y., died suddenly, April 10, 1931, while on a professional call. He was a graduate of the New York-American Veterinary College, class of 1906, and was associated in practice with Dr. W. H. Wheeler (N. Y. C. V. S. '98).

JAMES J. PATTERSON

Dr. James J. Patterson, of Seaford, Delaware, died May 4, 1931, while making a professional call. He was a graduate of the Grand Rapids Veterinary College, class of 1909, and had been in general practice at Seaford for over 20 years.

CLIFFORD E. BUTTS

Dr. Clifford E. Butts, of Gladbrook, Iowa, died May 15, 1931, following an automobile accident five days previously.

Born at Brooklyn, Iowa, July 7, 1894, Dr. Butts attended local grade and high schools before entering Iowa State College. He received his veterinary degree in 1917 and located at Brooklyn.

Dr. Butts joined the A. V. M. A. in 1919. He was also a member of the Iowa State Veterinary Association.

ALVIN R. LETSON

Dr. Alvin R. Letson, of Fountain, Mich., disappeared New Year's Day 1930, and it was believed, at the time, that he had met with foul play. On June 5, 1931, a body was recovered in the harbor at Manistee, Mich., and through a belt and shoe which remained on the decomposed body, it was identified as that of Dr. Letson. He was a graduate of the Grand Rapids Veterinary College, class of 1918, and was engaged in general practice at Fountain until his disappearance.

GEORGE W. CLIFFE

Dr. George W. Cliffe, of Upper Sandusky, Ohio, died June 7, 1931, aged 75 years, after a protracted illness. He was a graduate of the Ohio Veterinary College, class of 1892, and practiced at Upper Sandusky for almost forty years. He served two terms as sheriff of Wyandot County and made a splendid record as a law-enforcing officer.

Dr. Cliffe joined the A. V. M. A. in 1905 and served as resident secretary for Ohio, 1908-10. He was a member of the Ohio State Veterinary Medical Association and served as president for the term 1922-23.

WILLIAM O. McGUIGAN

Dr. William O. McGuigan, of East Liverpool, Ohio, aged 57, died June 14, 1931, in the City Hospital, after an illness of four months. He was a graduate of the Ontario Veterinary College, class of 1907, and practiced at Columbiana, Ohio, until about 1915, when he located at East Liverpool.

Dr. McGuigan was a member of the Ohio State Veterinary Medical Association. He is survived by a sister. Burial was at Cedar Springs, Ontario.

SAMUEL ESHLEMAN BRUNER

Dr. Samuel E. Bruner, of Camp Hill, Pa., died in the Harrisburg (Pa.) Hospital, June 25, 1931, following a brief illness. Dr. Bruner attended the 25th reunion of his class, at the University of Pennsylvania, Philadelphia, on June 12. He was taken sick that night and was unable to attend the alumni meeting the following day. He returned home on June 14 and entered the hospital on the 19th. Death was due to heart involvement with complications.

Following his graduation from the University of Pennsylvania, in 1906, Dr. Bruner practiced at Greensburg, Pa., for about eight years. He then accepted a position with the Pennsylvania Bureau of Animal Industry and was in charge of tuberculosis eradication work in the Keystone State when he severed his connection with the Bureau about eighteen months ago.

Dr. Bruner joined the A. V. M. A. in 1911. He was a member of the United States Live Stock Sanitary Association and the Pennsylvania State Veterinary Medical Association. Other affiliations included the Camp Hill Lodge F. and A. M.; Harrisburg Consistory, Scottish Rite Masons; Zembo Temple, Nobles of the Mystic Shrine; West Shore Country Club and Post 43, American Legion, of Camp Hill.

He is survived by his widow (née Mary Duttenhoffer), one son and one sister.

R. C. LIVINGSTON

Dr. R. C. Livingston, of Groton, S. Dak., aged 50, was killed in an automobile accident four miles north of Conde, June 19, 1931. Dr. Livingston was a registered non-graduate practitioner. He had attended the Chicago Veterinary College but failed to complete the course. He was a member of the Groton School Board. A wife, three daughters and a son survive.

G. P. McC.

Our sympathy goes out to Dr. and Mrs. B. C. Talley, of Bennettsville, S. C., in the death of Mrs. J. E. Colvert, mother of Mrs. Talley, at Statesville, N. C., May 17, 1931, of angina pectoris; and to Dr. William Herbert Lowe, of Paterson, N. J., in the death of his wife, Carrie Amiraux Lowe, June 7, 1931, after a protracted illness.

PERSONALS

MARRIAGES

Dr. J. H. Spurlock (K. S. C. '28) to Miss Margaret A. Feenane, both of Trenton, N. J., December 31, 1930, at Trenton, N. J.

Dr. Frank Thorp, Jr. (Iowa '26), of Urbana, Ill., to Miss Margaret Louis Plant, of Chicago, Ill., June 18, 1931, at Chicago, Ill.

PERSONALS

Dr. C. J. Price (K. S. C. '31) has located at Holdenville, Okla.

Dr. A. F. Eckert (Ont. '31) has located at Raymond, Ill., for general practice.

Dr. William C. Bowen (Ohio '31) will engage in general practice at North Lewisburg, Ohio.

Dr. T. J. Sharpe (St. Jos. '23), formerly of Coggan, Iowa, removed to Central City, Iowa, June 15.

Dr. Ira Dodson (Chi. '12) was recently appointed city veterinarian of Danville, Ill., by the city council.

Dr. Chas. H. Rosenstiel (Chi. '07) reports a change of address from Champaign, Ill., to Freeport, Ill., R. F. D. # 3.

Dr. G. A. Root (Ont. '06), who has practiced at Imlay City, Mich., for the past fourteen years, has removed to Yale, Mich.

Dr. Claude L. Lammey (U. P. '28) has resigned from the Bureau of Animal Industry to engage in general practice at York, Pa.

Dr. Peter H. Canakis (Chi. '16), who spent several months in Greece recently has returned to his home in Pierre, S. Dak.

Dr. Sam Crouch (McK. '20) has removed from Glendale, Calif., to Los Angeles and is now located at 436 San Fernando Rd.

Dr. W. Taylor Miller (K. S. C. '24) has reported a change of address from Ithaca, N. Y., to 3205 Morrison St. N. W., Washington, D. C.

Dr. O. J. Howard (Ind. '10), of Watervliet, Mich., is building a hospital for small animals and having his residence enlarged at the same time.

Dr. M. D. Moses (Chi. '16) has located at Sparta, Ill., for general practice. He was recently engaged in tuberculin testing in Randolph County.

Dr. L. E. Marbry (Ind. '10), of Centralia, Ill., has accepted an appointment as Bond County (Ill.) Veterinarian, with headquarters at Greenville, Ill.

Dr. M. B. Mertens (Chi. '18), of Mauston, Wis., who came to the United States from Holland, about twenty years ago, will visit his native land this summer.

Dr. C. A. Parkinson (Ont. '14), of Owosso, Mich., has announced his candidacy for one of the two vacancies on the School Board. The election will be held on July 13.

Dr. Glenn R. Dunning (Gr. Rap. '06), of Memphis, Mich., returned home early in June after spending two months in the Saint Joseph Sanitarium at Ann Arbor, Mich.

Dr. C. L. Barnes (Corn. '00) has been appointed Dutchess County (N. Y.) Veterinarian. He has his headquarters in the Farm Bureau Office, Post Office Bldg., Poughkeepsie, N. Y.

Dr. Lloyd D. Jones (Iowa '31) plans to enter general practice at Rochelle, Ill., in conjunction with his father who has been practicing in the same community for the past 32 years.

Dr. A. W. Groth (St. Jos. '20), of West Union, Iowa, recently accepted an appointment in the U. S. Bureau of Animal Industry, and has been assigned to meat inspection at New York.

Dr. E. A. McAtee (K. C. V. C. '10), of Barry, Ill., was painfully injured on June 15, while tagging cattle on a farm near Payson, Ill. He received fractures of several ribs on the left side.

Dr. J. F. Claire (Gr. Rap. '10), of Burbank, Calif., formerly with the Los Angeles County Live Stock Department, has located at Tracy, Calif., where he will engage in general practice.

Dr. Thomas W. White (San Fran. '12), of Malad, Idaho, was apponted to the position of director of the Bureau of Animal Industry, Idaho State Department of Agriculture, by Governor Ross, on June 6.

Dr. J. W. Berry (A. P. I. '23), of Pulaski, Tenn., was elected secretary of the Tennessee State Board of Veterinary Medical Examiners at the annual meeting of the Board, held in Nashville on June 10.

Dr. William M. Thomson (Corn. '16), formerly of the staff of the New York State Veterinary College at Cornell University, is now located at the Salamanca Veterinary Laboratory, 37 Atlantic St., Salamanca, N. Y.

Dr. R. E. Hammond (Mich. '22) has resigned his position as city veterinarian in the Bureau of Food Inspection, Division of Health, Dayton, Ohio, to accept a similar position with the city of Youngstown, Ohio.

Dr. T. J. Stearns (Chi. '16), of Russellville, Ky., has been appointed assistant state veterinarian by the State Live Stock Sanitary Board. He will be placed in charge of the Bourbon Stock Yards at Louisville, succeeding the late Dr. W. H. Simmons.

Dr. Charles E. Hagyard (Ont. '24), of Lexington, Ky., recently purchased the Greenridge Horse Farm, at public auction. Greenridge consists of 174 acres and Dr. Hagyard has announced that he would continue the farm as a Thoroughbred nursery.

Dr. F. L. Rogers (O. S. U. '26) has been given a provisional appointment as field veterinarian with the Ohio State Division of Animal Industry. Dr. Rogers will be engaged in work with the control of Bang's disease and will have his headquarters in Columbus.

Dr. Chauncey McCandless (Chi. '15), who served as state veterinarian of Ohio under Governor Cooper, has returned to Salem, Ohio, and will resume general practice there. During the past six months Dr. McCandless has been pursuing postgraduate studies at Ohio State University.

Dr. A. C. Spivey (Ind. '12), of Thorntown, Ind., has been appointed a member of the Indiana State Live Stock Sanitary Board, by Governor Leslie, succeeding Dr. C. E. Mummert (Ind. '07), formerly of Young America and now of Logansport, Ind. Dr. Spivey will take office July 8.

Dr. Adolph Eichhorn (N. Y.-Amer. '00), Director, Veterinary Department, Lederle Laboratories, Pearl River, N. Y., addressed the meeting of the New Jersey Health Officers' Association, at Jersey City, N. J., June 10, 1931, on the subject of "Transmission of Disease from Animal to Man." Practically every health officer in the state of New Jersey, to the number of approximately 300, was in attendance.

One of the many public schools in Kansas City.